

**In the United States Court of Federal Claims**  
**OFFICE OF SPECIAL MASTERS**  
**No. 17-917V**  
Filed: July 7, 2025

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RICHARD K. PARKER,

Petitioner,

v.

SECRETARY OF HEALTH AND  
HUMAN SERVICES,

Respondent.

\* \* \* \* \*

*Ramon Rodriguez III*, Sands Anderson PC, Richmond, VA, for Petitioner.

*Sarah Christina Duncan*, U.S. Department of Justice, Washington, DC, for Respondent.

**DECISION DENYING ENTITLEMENT<sup>1</sup>**

**Shah**, Special Master:

On July 7, 2017, Richard K. Parker (“Petitioner”) filed a petition for compensation under the National Vaccine Injury Compensation Program, 42 U.S.C. § 300aa-10, *et seq.*<sup>2</sup> (the “Vaccine Act” or “Program”). ECF No. 1 (“Pet.”). The petition alleges that Petitioner developed Parsonage-Turner syndrome (“PTS”) as a result of a pneumococcal 13-valent conjugate (“Prevnar 13”) vaccine he received on November 21, 2014. *Id.* at 1-2. The petition alternatively alleged that the subject vaccination significantly aggravated preexisting PTS. *Id.* at 2.

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<sup>1</sup> Because this Decision contains a reasoned explanation for the action in this case, it must be made publicly accessible and will be posted on the United States Court of Federal Claims’ website, and/or at <https://www.govinfo.gov/app/collection/uscourts/national/cofc>, in accordance with the E-Government Act of 2002. 44 U.S.C. § 3501 note (2018) (Federal Management and Promotion of Electronic Government Services). This means the Decision will be available to anyone with access to the internet. In accordance with Vaccine Rule 18(b), Petitioner has 14 days to identify and move to redact medical or other information, the disclosure of which would constitute an unwarranted invasion of privacy. If, upon review, I agree that the identified material fits within this definition, I will redact such material from public access.

<sup>2</sup> National Childhood Vaccine Injury Act of 1986, Pub. L. No. 99-660, 100 Stat. 3755. Hereinafter, for ease of citation, all “§” references to the Vaccine Act will be to the pertinent subparagraph of 42 U.S.C. § 300aa (2012).

For the reasons discussed in this decision, I find that Petitioner has failed to meet his burden of proof and is not entitled to compensation.

## **I. PROCEDURAL HISTORY**

After filing the petition, Petitioner filed affidavits and medical records. Exs. 1-10. On March 19, 2018, Respondent filed a Rule 4(c) Report recommending that entitlement be denied. ECF No. 18.

Petitioner filed additional medical records on April 30, 2018. Ex. 11. In January 2019, Petitioner filed an expert report from Lawrence Steinman, M.D., along with two versions of Dr. Steinman's curriculum vitae and medical literature. Exs. 12-43. On March 7, 2019, Respondent filed an expert report by Thomas P. Leist, M.D., Ph.D., along with Dr. Leist's curriculum vitae and medical literature. Exs. A & Tabs 1-3, B.

On April 3, 2019, former Special Master Katherine E. Oler directed Petitioner to file a supplemental expert report from Dr. Steinman addressing whether the proper diagnosis was PTS or a rotator cuff tear, whether a hypothetical onset of PTS up to 71 days after vaccination was medically appropriate, and other issues. ECF No. 35. Petitioner filed a second expert report and medical literature from Dr. Steinman on July 18, 2019. Exs. 47-51.

On September 24, 2019, Special Master Oler held a status conference at which she noted that there were "varying descriptions for the onset of shoulder pain." ECF No. 45 at 1. Special Master Oler informed the parties that where the record was inconsistent, the Court would err in favor of Petitioner. *Id.* She asked Petitioner to file additional evidence and respond to questions regarding onset. *Id.*

Petitioner filed additional evidence on November 22, 2019, including pre- and post-vaccination photographs and a supplemental affidavit. Exs. 52-60. On April 23, 2020, Respondent filed a second expert report from Dr. Leist and an expert report from Emanuel Maverakis, M.D., along with medical literature and Dr. Maverakis's curriculum vitae. Exs. C & Tabs 1-3, D & Tabs 1-18, E.

Petitioner filed a third expert report from Dr. Steinman on October 1, 2020. Ex. 61. On October 5, 2020, Petitioner filed an expert report from Daniel Carr, M.D., along with Dr. Carr's curriculum vitae. Exs. 62-63. On March 22, 2021, Respondent filed a third expert report from Dr. Leist, a second expert report from Dr. Maverakis, and an expert report from Geoffrey Abrams, M.D., along with medical literature and Dr. Abrams's curriculum vitae. Exs. F, G & Tabs 1-14, H & Tabs 1-20, I.

Special Master Oler scheduled an entitlement hearing for April 11-12, 2023. ECF No. 72. On February 2 and March 10, 2023, Petitioner filed additional medical records. Exs. 64-76. On March 14, 2023, Respondent filed additional medical literature. Exs. J-K. The parties filed prehearing briefs on March 14 and March 21, 2023. ECF Nos. 79, 85.

Special Master Oler conducted an entitlement hearing on April 11-12, 2023, at which Petitioner, his wife, and Drs. Steinman, Carr, Leist, Abrams, and Maverakis testified. After the hearing, the parties filed additional materials. Exs. 77, L. The parties filed post-hearing briefs on July 5, September 5, and November 7, 2023. EFC Nos. 106, 107, 109.

Both parties agreed that the record was complete on November 13, 2023. ECF No. 110. This case was reassigned to me on August 13, 2024. ECF No. 112. This matter is now ripe for adjudication.

## **II. FACT EVIDENCE**

Mr. Parker is a personal trainer and has lifted weights since he was 13 years old. Tr. at 29-30. Prior to the subject vaccination, he exercised and trained with weights regularly; his upper body routine included pyramids, skull crushers, dips, triceps curls, forearm curls, military presses, and more. *Id.* at 13-14. From 1985 to 1989, he used steroids on and off while weight training. *Id.* at 30-32. Although he had periodic rotator cuff injuries, which are “part of the trade” for weightlifters, he testified that he had no problems or pain in his right shoulder in the six months prior to vaccination. *Id.* at 12-15, 36.

Petitioner’s medical history prior to the subject vaccination was significant for lower back osteoarthritis and degenerative joint disease, skin cancer, diverticulosis, high blood pressure, high cholesterol, hypogonadism, hyperlipidemia, reflux esophagitis, left knee arthroscopy, and “[s]ensorimotor polyneuropathy involving the upper and lower extremities (worse in the lowers),” with “[s]uperimposed lower lumbar sacral motor radiculopathy being worse on the right side with active denervation components.” Ex. 4 at 1-16; Ex. 5 at 1-4; Ex. 6 at 1; Ex. 7 at 1-6.

Petitioner was 67 years old when he received a Prevnar 13 vaccination on November 21, 2014, at Kroger Pharmacy.<sup>3</sup> Ex. 8 at 1. Petitioner stated that the point of vaccination was “very sore” and that the vaccine “stung a little bit when it went in,” but he had no further immediate reaction. Tr. at 15, 37.

According to his first affidavit, executed on July 7, 2017, Petitioner began experiencing “pain and discomfort” in his neck, right shoulder, and right arm “in December 2014.” Ex. 1 (“Pet. Affidavit”) at 1. “By January 2015,” he noticed weakness in his right arm while weight training and “[b]y February 2015,” he noticed “obvious atrophy” in his right triceps and right pectoral area. *Id.* Petitioner observed that that when he held his right arm up, the skin on his right arm sagged from loss of muscle tone. *Id.* In a second affidavit executed November 21, 2019, Petitioner stated that after he received the vaccination, he continued weight training four days per week, four hours per session. Ex. 60 (“Pet. Second Affidavit”) at 1.

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<sup>3</sup> Although the medical records do not indicate the arm in which Petitioner received the vaccination, Petitioner and Mrs. Kyndall Parker testified that Petitioner received the vaccine in his right arm. Tr. at 7, 45. Petitioner attempted to obtain records from Kroger Pharmacy indicating the site of administration but was advised that no such records exist. Ex. 72.

At the hearing, Petitioner testified that he noticed pain, soreness, and stiffness in his neck and shoulder on December 20 or 21, 2014; however, during cross-examination he could not explain how he remembered those specific dates, and he acknowledged that he did not report those dates to his treating physicians in 2015. Tr. at 8, 39-40. He testified that at the beginning of January 2015, weakness and atrophy began to develop in his right triceps, shoulder, and pectoral muscle. *Id.* at 15-16. He could no longer continue with his normal workout routine; he could only lift half the weight, his right arm would fail, and he could no longer balance free weights. *Id.* at 15.

Petitioner's wife, Mrs. Kyndall Parker, submitted an affidavit executed July 4, 2017. Ex. 2 ("Mrs. Parker Affidavit"). Mrs. Parker stated that "around the end of December 2014," Petitioner was "noticeably uncomfortable" on the right side of his chest, right arm, and right shoulder. *Id.* at 1. During the hearing, Mrs. Parker testified that within "two to three weeks" of vaccination, Petitioner "started complaining of having shoulder pain," as well as pain in his neck and down his right arm. Tr. at 45. Mrs. Parker suggested that Petitioner call his physician, but he decided to "wait until the end of December" to see if the pain resolved on its own. *Id.*

Mrs. Parker stated that "[b]y January 2015," Petitioner noticed "a lot of weakness in his right arm when he was exercising." Mrs. Parker Affidavit at 1. In January, he attempted to schedule an appointment with James R. Dageforde, M.D., his primary care physician ("PCP") of 20 years, but was advised Dr. Dageforde was out for knee surgery. Tr. at 9, 46; Pet. Affidavit at 1. He tried calling Dr. Dageforde again at the end of February, but Dr. Dageforde was still out on medical leave and was booked until April. Pet. Affidavit at 1; Mrs. Parker Affidavit at 1.

Mrs. Parker testified that while Petitioner was waiting for his initial appointment with Dr. Dageforde, "we had just kind of looked to see what some of the possibilities could be because he never had any of these symptoms before," and they came to believe that Petitioner had PTS. Tr. at 68. Mrs. Parker also discussed Petitioner's condition with friends who were nurses, and they too believed that he had PTS. *Id.*

According to the medical records, Petitioner presented to Dr. Dageforde on April 20, 2015, for a testosterone injection, his yearly follow-up on hypertension and hyperlipidemia, and atrophy in "portions of the right biceps and right pectoral muscles." Ex. 7 at 8. Petitioner testified that he showed Dr. Dageforde the atrophy in his right arm and sagging in his right pectoral muscle. Tr. at 11. Dr. Dageforde noted that Petitioner felt "like he might have [PTS]." Ex. 7 at 8. On exam, Dr. Dageforde observed atrophy in the lateral right pectoral muscle and right triceps. *Id.* at 8-9. His assessment was "arm muscle atrophy," and he noted: "I'm not sure what the cause is. I think he does need a referral to a neurologist." *Id.* at 9.

On May 28, 2015, Petitioner saw Daniel M. Hardy, M.D., a neurologist, for "[w]asting of muscle right tricep[s] and partial pectoral." Ex. 5 at 5. Petitioner reported that after the subject vaccination, "he developed some discomfort in his neck," which started in January 2015. *Id.* "Over the subsequent months, he began to notice atrophy in his right triceps and pectoral muscle as well as weakness when he was doing his ordinary weightlifting." *Id.* Petitioner did not repeat his report to Dr. Dageforde that he had experienced right biceps atrophy.

Dr. Hardy noted that Petitioner was “an avid weightlifter and ha[d] extremely hypertrophied muscles throughout his entire upper body,” such that “[t]he atrophy in his right triceps and pectoral muscles [was] extremely obvious.” Ex. 5 at 5. On exam, Petitioner’s motor strength was “5/5 in all 4 extremities except [right] tricep[s] and pectoral muscle which show[ed] weakness and atrophy[,]” and his range of motion (“ROM”) was normal. *Id.* at 6. Dr. Hardy diagnosed Petitioner with PTS. *Id.* He did not feel further testing was needed to confirm this diagnosis. *Id.*

Petitioner testified that Dr. Hardy told him his Prevnar 13 vaccination was the cause of his PTS. Tr. at 18. Petitioner’s affidavit also states that Dr. Hardy “confirmed the diagnosis of [PTS] and felt the Prevnar 13 vaccine was the cause.” Pet. Affidavit at 2. Similarly, Mrs. Parker’s affidavit states that Dr. Hardy confirmed Petitioner’s PTS diagnosis and “believed the cause to be the Prevnar-13 vaccination.” Mrs. Parker Affidavit at 1-2. According to the medical records, however, Dr. Hardy commented only that Petitioner’s PTS “followed a few months after [his] pneumonia vaccine in that arm.” Ex. 5 at 6. Dr. Hardy informed Petitioner that “he could expect gradual improvement over the next 6-12 months” and that “there [was] nothing he can really do that will make this improve more or faster.” *Id.* Petitioner reported that was “already starting to see some mild improvement.” *Id.*

On August 12, 2015, Petitioner presented to Matthew H. Walker, M.D., an orthopedist, for what he thought was a rotator cuff problem. Tr. at 22, 34; Ex. 9 at 1-5. His chief complaint was “right-sided shoulder pain” that had bothered him for “about six months.” Ex. 9 at 1. He reported that he did not “recall any particular injury” to his shoulder, that six months earlier “he was diagnosed with [PTS] on the right side,” and that he felt that his “whole right upper extremity [had] gotten weak over the past six months.”<sup>4</sup> *Id.*

On exam, Petitioner had no radicular symptoms and had normal sensation. Ex. 9 at 1. He had no tenderness to palpation over the acromioclavicular (“AC”) joint. *Id.* He was “a little sore over the biceps tendon” and had some decreased strength in the bilateral external rotators. *Id.* X-rays revealed “a little proximal humeral migration” and “some AC joint arthrosis.” *Id.* Dr. Walker assessed some weakness in the rotator cuff, but he noted that it was difficult to say if it was “from his [PTS] or from his rotator cuff tear.” *Id.* He was “more concerned about [a] rotator cuff tear than the [PTS]” and ordered an MRI for further evaluation. *Id.*

Petitioner underwent an MRI on September 9, 2015, which showed rotator cuff tears, AC joint arthropathy, and a superior labrum anterior and posterior (“SLAP”) tear. Ex. 9 at 6-7.

On September 30, 2015, Dr. Walker advised Petitioner that the MRI revealed an approximately 1.5-cm “full thickness rotator cuff tear.” Ex. 9 at 8. He recommended arthroscopic

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<sup>4</sup> Notably, a patient history completed the same day at Dr. Walker’s office reflected varying onset dates of Petitioner’s symptoms. The “Patient Information” section of the record reported an injury onset date of June 1, 2015, while the “General History” section reported an onset date of December 1, 2014. Ex. 9 at 2, 4. The record also indicates that Petitioner received a pneumococcal vaccine in October 2014 (the actual date of the Prevnar 13 vaccination was November 21, 2014) and an influenza vaccine in November 2014. *Id.* at 5.

repair. *Id.* Surgery would include a “lengthy recovery period,” requiring Petitioner to wear a sling for six weeks and devote a year to regaining his strength and motion. *Id.* Petitioner declined to have surgery and sought a second opinion. Tr. at 24-25; 55-56.

On January 11, 2016, Petitioner saw orthopedist Geoffrey Higgs, M.D., for right shoulder pain and to follow up on his 2011 left knee arthroscopy, which Dr. Higgs had performed. Ex. 4 at 19. He reported that his right shoulder pain had been present since July 2015 (not December 2014, as he testified) and that he had PTS. *Id.* He stated that “the pain [was] localized to the right lateral shoulder with lateral movements” and that he was experiencing intermittent numbness and tingling, as well as cracking and popping. *Id.* He denied radicular symptoms. *Id.* On exam, he had significant triceps atrophy and positive Hawkins, Neer, empty can, and O’Brien’s tests. *Id.* ROM of the right shoulder was restricted. *Id.* Scapulothoracic dyskinesis<sup>5</sup> was present. *Id.* He had 4/5 strength on external rotation and forward elevation and 5/5 strength on internal rotation. *Id.* No muscle atrophy or gross deformities were observed. *Id.*

Dr. Higgs’s assessment was a right rotator cuff tear. Ex. 4 at 20. He commented that “[t]he question is whether [Petitioner’s] weakness is due to a large rotator cuff tear or an overlap from the [PTS].” *Id.* He planned to evaluate the MRI imaging, “which supposedly only show[ed] a small tear,” and he remarked that if Petitioner in fact had only a small tear, then it would be reasonable for him to continue working out and performing his usual activities as tolerated. *Id.*

Mr. and Mrs. Parker testified that Dr. Higgs also recommended physical therapy (“PT”) to treat the rotator cuff tear. Tr. at 26, 58. Petitioner presented to PT on January 13, 2016, describing an eight-month history of “[s]capulothoracic atrophy and dysfunction” due to his PTS diagnosis and rotator cuff wasting. Ex. 6 at 6. Over the next month, Petitioner attended four sessions of PT, which he testified improved his symptoms. *Id.* at 14, 16, 20; Tr. at 33. Mrs. Parker testified that Petitioner said his physical therapist identified a small divot in the right lower portion of his thumb and forefinger, which he characterized as a sign of PTS. Tr. at 58-59.

On February 19, 2016, Petitioner followed up with Dr. Hardy “at the request of his lawyer to facilitate a settlement.” Ex. 5 at 8. Petitioner’s examination was unchanged from his May 28, 2015 visit. *Id.* at 6, 9. Mrs. Parker testified that during Petitioner’s exam, Dr. Hardy noted the same divot that Petitioner’s physical therapist identified; that was not documented in the medical record. Tr. at 59-61; *see* Ex. 5 at 9. Dr. Hardy’s assessment was that Petitioner was “significantly improved from when I saw him in May 2015, but his syndrome remains present.” Ex. 5 at 9. Additionally, Dr. Hardy noted that Petitioner would continue to gradually improve, and, although he was unlikely to “return to 100% of his baseline, he will likely not be far off.” *Id.*

On April 6, 2016, Petitioner followed up with Dr. Dageforde and reported that he saw a neurologist for PTS that developed after the subject vaccination. Ex. 7 at 11. Dr. Dageforde

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<sup>5</sup> Scapulothoracic: pertaining to the scapula and thorax. DORLAND’S, <https://www.dorlandsonline.com/dorland/definition?id=44767&searchterm=scapulothoracic> (last visited June 30, 2025); Dyskinesia: distortion or impairment of voluntary movement, as in tic, spasm, or myoclonus. DORLAND’S, <https://www.dorlandsonline.com/dorland/definition?id=15221&searchterm=dyskinesia> (last visited June 30, 2025).

treated Petitioner for several chronic conditions. *Id.* at 12. No specific exam or treatment for PTS or rotator cuff tear was provided. *Id.* at 11-12.

Petitioner returned to PT on August 3, 2016, for “shoulder pain and weakness that [was] recently exacerbated by overhead weightlifting.” Ex. 6 at 42. Over the next month, Petitioner attended eight PT sessions. Ex. 6 at 39-41.

Petitioner presented to R. Ward Morgan, M.D., a new PCP, on February 16, 2017, for a routine checkup. Ex. 7 at 17. Petitioner’s medical history included “[PTS] - after Prevnar 13 vaccine - seen by neurology.” *Id.* He declined an influenza vaccination. *Id.* at 19. Dr. Morgan’s exam, assessment, and treatment did not address purported PTS. *Id.* at 17-19.

On September 27, 2017, Petitioner presented to John Mills, D.O., another new PCP, for a “torn R shoulder rotator cuff,” low testosterone, a bump on his neck, and right-hand issues. Ex. 66 at 4. Dr. Mills’s exam, assessment, and treatment did not address Petitioner’s right rotator cuff. *Id.* at 4-5.

Seven months later, on April 17, 2018, Petitioner had a cervical spine CT scan prompted by his right shoulder pain. Ex. 66 at 21. The exam revealed “[e]xtensive degenerative disc disease with disc space narrowing and osteophyte[s] at C3-C4, C4-C5, C5-C6, C6-C7, and C7-T1 as well as T1-T2,” in addition to “subtle degenerative anterolisthesis of C7 on T1.” *Id.*

Petitioner returned to neurologist Dr. Hardy on February 1, 2019, following an emergency room (“ER”) visit the prior December. Ex. 67 at 1. Petitioner reported that while he was working out at the gym, he “stood up abruptly from plank position and became lightheaded with a brief syncope.” *Id.* Dr. Hardy noted that he had seen Petitioner “in 2016 for [PTS] caused by a pneumonia vaccine. Litigation for this is still ongoing.” *Id.* Dr. Hardy concluded that the syncopal episode was consistent with “an episode of orthostatic hypotension leading to syncope” and advised Petitioner to “take care when he is changing positions abruptly.” *Id.* at 2.

On September 15, 2020, Petitioner presented to neurosurgery nurse practitioner Brittany Henderson for lower extremity weakness. Ex. 68 at 1. Petitioner reported that over the previous two years he had developed recurrent, progressive bilateral leg weakness that caused “significant troubles getting up from a seated position” and in going up and down stairs. *Id.* In his medical history, he reported PTS following a vaccination in 2014. *Id.* On exam, Petitioner’s strength was normal in all muscles. *Id.* at 2. An x-ray taken that day revealed multilevel degenerative disc disease. *Id.* at 3. Nurse Henderson ordered an MRI of the lumbar spine and referred Petitioner to PT. *Id.*

From September 21 to October 1, 2020, Petitioner attended four PT sessions for lower back pain. Ex. 70 at 1, 14. On October 19, 2020, Petitioner was discharged from PT with moderate improvement but without meeting his goals. *Id.* at 12.

Petitioner presented to Dr. Mills on April 14, 2021, to discuss ordering a chair lift for stairs and a walk-in tub, necessitated by his upper and lower extremity weakness from PTS and lumbar spinal stenosis. Ex. 66 at 16. Petitioner reported that he was “[e]ssentially unable to go to the gym

anymore” and that he was “getting by with a cane” but “refuses to use a walker.” *Id.* Additionally, Petitioner reported that it was “[v]ery difficult to get out of a chair,” requiring multiple attempts, and that stairs were even more of a problem due to his leg weakness. *Id.* On exam, Dr. Mills noted “[d]iffuse weakness” in both arms, shoulders, and legs. *Id.* He observed that it took multiple attempts for Petitioner to get out of a chair and that he walked slowly with a cane and a stooped posture. *Id.* Dr. Mills’s assessment included spinal stenosis in the lumbar region, lumbar disc disease, PTS, bilateral leg and arm weakness, and hypertension. *Id.* at 16-17. Dr. Mills noted that Petitioner would benefit from a lift chair, stair lift, and walk-in tub due to his spinal stenosis and PTS. *Id.* at 17.

On September 20, 2021, Petitioner saw Allison McInnis, P.A., for lower back pain. Ex. 69 at 6. Petitioner reported worsening bilateral leg weakness and decreasing balance, as well as heaviness and fatigue in his legs when standing and walking. *Id.* He said he was able to walk approximately two blocks before stopping to rest his legs. *Id.* He also reported “numbness and tingling in his fingers bilaterally,” causing him to drop objects. *Id.* In his medical history, Petitioner reported PTS and noted that it affected his right hand. *Id.* P.A. McInnis recommended bilateral L2-3 epidural steroid injections, which Petitioner deferred, along with an MRI of the cervical spine. *Id.* An October 13, 2021 cervical spine MRI revealed “evidence of focal myelomalacia at C6 of indeterminate origin” and “diffuse degenerative disc disease and facet arthritis with relatively mild narrowing of the canal at multiple levels.” Ex. 66 at 27-29.

On November 2, 2021, Petitioner followed up with P.A. McInnis to discuss the results of his cervical spine MRI. Ex. 69 at 3. P.A. McInnis noted that Petitioner had “symptoms of early cervical myelopathy with a focal finding of myelomalacia on his cervical MRI at C6.” *Id.* at 5. P.A. McInnis recommended that Petitioner “see Dr. Van<sup>6</sup> for further evaluation of possible surgical need for his cervical spine.” *Id.*

Petitioner returned to Dr. Mills on April 6, 2022, for a medication check and with “[q]uestions about a scooter for mobility.” Ex. 74 at 4. Dr. Mills noted that Petitioner was “in a wheelchair and has a cane for assistance.” *Id.* at 5. On exam, Dr. Mills noted that Petitioner needed to “rock several times to stand up out of a chair” and that he had edema in his ankles and distal lower extremities. *Id.* He said he would “look into the possibility of a scooter” and referred Petitioner for an EMG/NCV of the legs. *Id.* PTS was included in Dr. Mills’s assessment but was not otherwise addressed. *Id.* Petitioner was noted to have had three doses of the Moderna COVID vaccine on January 22, February 22, and October 30, 2021. *Id.* at 6.

Petitioner presented to Dr. Mills on December 8, 2022, for a telehealth visit to “discuss [r]ehab” following several hospitalizations over two and a half months.<sup>7</sup> Ex. 74 at 8. Petitioner had been hospitalized in September 2022 with Covid pneumonia. *Id.* He improved and was discharged, but he declined inpatient rehabilitation and did not do well at home, causing him to be readmitted to the hospital. *Id.* In October, he was discharged to rehab and eventually sent home. *Id.* Not long thereafter, however, he worsened again and was hospitalized with Proteus bacteremia.

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<sup>6</sup> Petitioner did not file medical records from Dr. Van.

<sup>7</sup> Petitioner did not file any records of these hospitalizations or rehabilitation stays.

*Id.* He was later sent to another rehabilitation facility, and he was discharged home on November 18, 2022. *Id.* Dr. Mills noted that Petitioner was having home health PT “with varying degrees of success.” *Id.*

Dr. Mills commented that during one of Petitioner’s hospital stays, he was diagnosed with a cerebellar cardiovascular accident (“CVA”). Ex. 74 at 8, 37-38. Petitioner’s current issues were debility, ataxia, tremor, neuropathy, and fall risk. *Id.* at 8. Dr. Mills referred Petitioner to outpatient stroke rehab. *Id.* at 9.

Petitioner saw Dr. Hardy on December 27, 2022, and described his hospitalizations. Ex. 75 at 1. Dr. Hardy noted that he reviewed Petitioner’s October 26, 2022 head CT and agreed that it showed a cerebellar infarct that had been present since at least the previous year. *Id.* He also noted that after Petitioner was discharged from rehab in November, he remained wheelchair-bound because he felt shaky when walking. *Id.* He was starting PT that week. *Id.* Dr. Hardy encouraged Petitioner to “participate fully with physical therapy” and diagnosed him with essential tremor. *Id.* at 3.

On January 20, 2023, Petitioner presented to Dr. Sujoy Gill, M.D., a pulmonologist, for shortness of breath. Ex. 76 at 3. Dr. Gill diagnosed Petitioner with dyspnea post-Covid with “very poor” spirometry and obesity. *Id.* at 4. He prescribed albuterol and provided educational materials on diet. *Id.*

There are no additional medical records after January 2023. At the April 2023 entitlement hearing, Petitioner testified that he still had atrophy in his right pectoral and right triceps muscles and that his “strength is still not what it used to be.” Tr. at 37-38.

### **III. EXPERT EVIDENCE**

#### **A. Expert Reports**

##### **1. Lawrence Steinman, M.D.: First Expert Report**

Dr. Steinman submitted three reports in this case. Ex. 12 (“First Steinman Rep.”); Ex. 47 (“Second Steinman Rep.”); Ex. 61 (“Third Steinman Rep.”). He also testified at the entitlement hearing. Tr. at 71-117; 293-311.

Dr. Steinman earned his M.D. from Harvard Medical School in 1973 and is board certified in neurology. Ex. 14 (“Steinman CV”) at 1; Tr. at 71, 73. Dr. Steinman has taught neurology, pediatrics, and genetics since 1980. Steinman CV at 1; Tr. at 71. He is the GA Zimmerman Chaired Professor of Neurology, Neurological Sciences, and Pediatrics at Stanford University and the former chairman of the university’s immunology department. Steinman CV at 1; Tr. at 71.

In 2009, Dr. Steinman was elected to the National Academy of Medicine, and in 2015 he was elected to the National Academy of Sciences. Steinman CV at 2; Tr. at 73. He has published more than 500 peer-reviewed papers and receives “calls from all over the world on a regular basis”

for outpatient consultations. Steinman CV at 5-46; Tr. at 73. During 43 years of clinical practice, he has seen approximately 20 patients with PTS. Tr. at 72.

Based on the medical records and the opinion of Dr. Hardy, Dr. Steinman concluded that Petitioner had PTS. First Steinman Rep. at 5. The Vaccine Injury Table (“Table”) characterizes brachial neuritis/PTS as follows:

[D]ysfunction limited to the upper extremity nerve plexus (i.e., its trunks, divisions, or cords). A deep, steady, often severe aching pain in the shoulder and upper arm usually heralds onset of the condition. The pain is typically followed in days or weeks by weakness in the affected upper extremity muscle groups. Sensory loss may accompany the motor deficits, but is generally a less notable clinical feature. Atrophy of the affected muscles may occur. The neuritis, or plexopathy, may be present on the same side or on the side opposite the injection. It is sometimes bilateral, affecting both upper extremities.

42 C.F.R. § 100.3(c)(6). Dr. Steinman opined that the Table definition of PTS “perfectly fits Petitioner’s case.” First Steinman Rep. at 5.

Dr. Steinman set forth two theories explaining how the Prevnar 13 vaccine could have triggered PTS in Petitioner. First Steinman Rep. at 6-15. His first theory was based on molecular mimicry, which involves “shared structures on a virus or bacteria or in a vaccine [that] can trigger a cross-reactive response to self,” leading to disease. *Id.* at 6. Specifically, he opined that the Prevnar 13 vaccine contains molecular mimics of phospholipids in peripheral nerve myelin that are involved in PTS. See Tr. at 79-80.

Dr. Steinman acknowledged that “[t]he immunology of [PTS] has not been studied in great detail.” First Steinman Rep. at 7. He cited two animal studies that suggest the disease is caused by an autoimmune response to components of peripheral nerve myelin. *Id.* In Wisniewski, the authors induced recurrent experimental allergic ganglioradiculoneuritis, which is “akin to PTS,” in monkeys injected with peripheral nerve myelin. Henryk M. Wisniewski et al., *Recurrent Experimental Allergic Polyganglioradiculoneuritis*, 30 ARCH NEUROL 347 (1974) (Ex. 25) (“Wisniewski”). In Brostoff, “guinea pigs developed inflammation in their brachial plexus, though no clinical symptoms, following injections of brachial plexus myelin.” First Steinman Rep. at 7 (citing S.W. Brostoff et al., *Immunopathologic response in guinea pigs sensitized with peripheral nervous system myelin*, 58 BRAIN RSCH. 500 (1973) (Ex. 26) (“Brostoff”)).

Dr. Steinman further opined that phospholipids present in the myelin sheath are a specific target in inflammatory neuropathies of both the peripheral and central nervous systems. First Steinman Rep. at 7-8. Antibodies to phospholipids are seen in patients with peripheral inflammatory neuropathies, such as Guillain-Barré syndrome (“GBS”). *Id.* at 7 (citing B. Gilburd et al., *Autoantibodies to Phospholipids and Brain Extract in Patients with the Guillain-Barré Syndrome: Cross-Reactive or Pathogenic?*, 16 AUTOIMMUNITY 23 (1993) (Ex. 27) (“Gilburd”)). Dr. Steinman analogized PTS to GBS, stating that PTS “can be likened to a localized version of

inflammatory neuropathy.” *Id.* The antibodies seen in GBS patients target the polar head group of the phospholipids phosphatidyl-choline, phosphatidylserine, and phosphatidyl-ethanolamine. *Id.* at 8 (citing Gilburd).

Similarly, Dr. Steinman’s research has shown that phospholipids are targeted in inflammation of the central nervous system. First Steinman Rep. at 8 (citing Jennifer L. Kanter et al., *Lipid Microarrays Identify Key Mediators of Autoimmune Brain Inflammation*, 12 NATURE MED. 138 (2005) (Ex. 28) (“Kanter”); Peggy P. Ho et al., *Identification of Naturally Occurring Fatty Acids of the Myelin Sheath That Resolve Neuroinflammation*, 4 SCI. TRANSLATIONAL MED. 1 (2012) (Ex. 29) (“Ho”)). In Ho, for example, autoantibodies to a phosphate group in phosphatidylserine and oxidized phosphatidylcholine derivatives were seen in an animal model of multiple sclerosis. *Id.* Taken together, these studies show evidence of an antibody response to phosphatidylcholine structures in inflammatory neuropathies. *Id.*

Next, Dr. Steinman opined that phospholipids are present in Prevnar 13 vaccine.<sup>8</sup> First Steinman Rep. at 8. For example, in the 19A component of Prevnar 13 there is expression of phosphorylcholine. *Id.* at 9 (citing Yi-Ping Chuang et al., *Impact of the glpQ2 Gene on Virulence in a Streptococcus pneumoniae Serotype 19A Sequence Type 320 Strain*, 83 INFECTION & IMMUNITY 682 (2015) (Ex. 30) (“Chaung”); K. Aaron Geno et al., *Pneumococcal Capsules and Their Types: Past, Present, and Future*, 28 CLINICAL MICROBIOLOGY REV. 871 (2015) (Ex. 32) (“Geno”)). This phospholipid “plays a key role in the pathophysiology of the pneumococcal pneumonia and the infection . . . [I]t would be logical therefore that a vaccine like Prevnar 13 would immunize the recipient against this component of pneumococcus.” *Id.* Also, the enzyme for producing phosphorylcholine is present in several antigen strains present in the Prevnar 13 vaccine. *Id.* at 10. Furthermore, the pneumococcal polysaccharides contained in the vaccine are complex and allow for the chemical attachment of phospholipids “via the glycerol moiety.” *Id.* at 10-11.

Dr. Steinman attempted to obtain additional information on the chemistry of the Prevnar 13 vaccine through inquiries to the Centers for Disease Control (“CDC”). First Steinman Rep. at 12. But the CDC declined to provide information on the specific chemistry of the vaccine, noting that it was proprietary to the manufacturer. *Id.* Based on the information available to him, Dr. Steinman concluded that Prevnar 13 “contains phospholipids that are targeted by the immune system in inflammatory neuropathy. PTS is a localized version of inflammatory neuropathy.” *Id.* at 13.

The second component of Dr. Steinman’s theory was that the aluminum adjuvant in the Prevnar 13 vaccine can cause PTS. First Steinman Rep. at 13-15. The adjuvant activates the Nalp3 inflammasome in the immune system and produces the pro-inflammatory cytokines interleukin-1 (“IL-1”) and interleukin-18 (“IL-18”). *Id.* at 13 (citing Stephanie C. Eisenbarth et al., *Crucial role for the Nalp3 inflammasome in the immunostimulatory properties of aluminum adjuvants*, 453

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<sup>8</sup> According to its package insert, the Prevnar 13 vaccine contains saccharides of 13 of the capsular antigens of the *Streptococcus pneumoniae* bacterium, conjugated to the non-toxic diphtheria CRM<sub>197</sub> protein. First Steinman Rep. at 6 (citing Ex. 22 (Prevnr 13 Prescribing Information)). The vaccine also contains an aluminum adjuvant. *Id.*

NATURE 1122 (2008) (Ex. 33) (“Eisenbarth”); Anna Sokolovska et al., *Activation of Dendritic cells and induction of CD4 T cell differentiation by aluminum containing adjuvants*, 25 VACCINE 4575 (2007) (Ex. 34) (“Sokolovska”); J.W. Mannhalter et al., *Modulation of the human immune response by the non-toxic and nonpyrogenic adjuvant aluminum hydroxide: effect on antigen uptake and antigen presentation*, 61 CLIN. EXP. IMMUNOL. 143 (1985) (Ex. 35) (“Mannhalter”); Hanfen Li et al., *Aluminum Hydroxide Adjuvants Activate Caspase-1 and Induce IL-1 $\beta$  and IL-18 Release*, 178 J. IMMUNOL. 5271 (2007) (Ex. 36) (“Li”)).

Although there are no studies on IL-1 $\beta$  or IL-18 in brachial neuritis, Dr. Steinman noted that “IL-1 and IL-18 are strongly upregulated during active GBS and its animal models and [are] reduced as GBS resolves.” First Steinman Rep. at 13-14. He argued that a translated abstract of a review of PTS cases reported clinical similarities between PTS and GBS; it does not appear, however, that that abstract was filed.<sup>9</sup> *Id.* at 13. Dr. Steinman cited other human and animal studies of GBS that he opined “constitute a strong scientific foundation for providing a basis for how the Prevnar 13 vaccine can induce PTS.” *Id.* at 15 (citing Mannhalter; Li; Kishan K. Nyati et al., *Correlation of Matrix Metalloproteinases-2 and -9 with Proinflammatory Cytokines in Guillain-Barre Syndrome*, 88 J. OF NEUROSCIENCE RSCH. 3540 (2010) (Ex. 37) (“Nyati”); Sebastian Jander & Guido Stoll, *Interleukin-18 is induced in acute inflammatory demyelinating polyneuropathy*, 114 J. OF NEUROIMMUNOLOGY 253 (2001) (Ex. 38) (“Jander & Stoll”); Shou Yu et al., *Neutralizing Antibodies to IL-18 Ameliorate Experimental Autoimmune Neuritis by Counter-Regulation of Autoreactive Th1 Responses to Peripheral Myelin Antigen*, 61 J. OF NEUROPATHOLOGY & EXPERIMENTAL NEUROLOGY 614 (2002) (Ex. 39) (“Yu”); Duan; Lawrence Steinman, *Some Misconceptions about Understanding Autoimmunity through Experiments with Knockouts*, 185 J. OF EXPERIMENTAL MED. 2039 (1997) (Ex. 41) (“L. Steinman”); B. Sun et al., *Passive Transfer of Experimental Autoimmune Neuritis by IL-12 and IL-18 Synergistically Potentiated Lymphoid Cells is Regulated by NKR-P1+ Cells*, 65 SCANDINAVIAN J. OF IMMUNOLOGY 412 (2007) (Ex. 42) (“Sun”); Lawrence B. Schonberger et al., *Guillain Barre Syndrome following vaccination in the National Influenza Immunization Program*, 110 AM. J. OF EPIDEMIOLOGY 105 (1979) (Ex. 43) (“Schonberger”)).

Lastly, Dr. Steinman noted that his theory for how Prevnar 13 vaccine could cause PTS is “the exact same” as the theory underpinning the Table injury of PTS following tetanus vaccination. First Steinman Rep. at 15.

With respect to onset, Dr. Steinman noted that, in addition to PTS, Petitioner was diagnosed with a rotator cuff tear by Drs. Higgs and Walker. First Steinman Rep. at 5. With two conditions present, the key issue is the timing of the development of PTS and of the rotator cuff tear. *Id.* Based on Dr. Hardy’s notes from Petitioner’s May 28, 2015 appointment, in which Petitioner reported symptoms beginning “in January [2015],” Dr. Steinman placed the onset of PTS at a minimum of 41 days after the immunization. *Id.* (citing Ex. 5 at 6). He did not specify a date of

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<sup>9</sup> Dr. Steinman referenced Rui-Sheng Duan et al., *IL-18 deficiency inhibits both Th1 and Th2 cytokine production but not the clinical symptoms in experimental autoimmune neuritis*, 183 J. NEUROIMMUNOLOGY 162 (2007) (Ex. 40) (“Duan”)), in discussing the “abstract” delineating the clinical similarities between PTS and GBS. First Steinman Rep. at 13-14. Duan, however, was an animal study that did not address PTS or GBS.

onset of the rotator cuff tear. Dr. Steinman opined that “[b]ased on a surrogate for GBS after influenza vaccination,” the onset of PTS “within about 41 days fits a well-established temporal interval.” *Id.* at 15-16 (citing).

## 2. Thomas P. Leist, M.D., Ph.D.: First Expert Report

Dr. Leist submitted three reports in this case. Ex. A (“First Leist Rep.”); Ex. C (“Second Leist Rep.”); Ex. F (“Third Leist Rep.”). He also testified at the entitlement hearing. Tr. at 142-80.

Dr. Leist earned his M.D. from the University of Miami and his Ph.D. in biochemistry from the University of Zurich. Ex. B (“Leist CV”) at 1; Tr. at 142. He is licensed in Pennsylvania, Maryland, and New York and is board certified in adult neurology by the American Board of Psychiatry and Neurology. Leist CV at 1; Tr. at 143. Currently, Dr. Leist is a Professor of Neurology, Division Chief of Clinical Neuroimmunology, and Director of the Comprehensive Multiple Sclerosis Center at Thomas Jefferson University. Leist CV at 1; Tr. at 144. He is also a member of the American Academy of Neurology, serves on the editorial board for *Practical Neurology* and *Frontiers in Neurology*, and is an ad-hoc reviewer for several journals, including the *Journal of Neuroimmunology* and *Lancet Neurology*. Leist CV at 2; Tr. at 143. Over his 20-year career at Thomas Jefferson University, Dr. Leist has seen between 15 and 20 cases of PTS. Tr. at 145-46.

Dr. Leist opined that Petitioner did not have PTS; rather, his symptoms were “due to a right rotator cuff tear/shoulder problem.” First Leist Rep. at 6. According to Feinberg & Radecki,

[t]he classic description of PTS is a condition in which the patient initially and suddenly develops constant, severe unilateral shoulder girdle pain . . . The duration of pain is almost always self-limiting, lasting 1 to 2 weeks, but on rare occasion persisting for longer periods of time. Although not present initially, weakness may develop a few days to weeks after the initial onset of symptoms.

Joseph H. Feinberg & Jeffrey Radecki, *Parsonage-Turner Syndrome*, 6 HHS J. 199 (2010) (Ex. A, Tab 1) (“Feinberg & Radecki”); First Leist Rep. at 5-6.

Here, Petitioner reported a history of about six months of right shoulder pain at his August 12, 2015 appointment with orthopedist Dr. Walker, placing the onset of his shoulder symptoms in late January or February 2015. First Leist Rep. at 6; Ex. 9 at 1. Dr. Leist opined that Petitioner’s “history of chronic shoulder pain is not consistent for [PTS],” and as Dr. Walker suspected, a September 9, 2015 MRI confirmed Petitioner had a right rotator cuff tear. First Leist Rep. at 6; Ex. 9 at 6. Further, Dr. Dageforde noted at his April 20, 2015 appointment that Petitioner had “arm muscle atrophy.” Ex. 7 at 9. Dr. Leist explained that “[i]t is known that rotator cuff tears can cause atrophy of muscles of the shoulder girdle including the biceps muscle.” First Leist Rep. at 6 (citing Eiichiro Iwata et al., *Biceps-Related Physical Findings Are Useful to Prevent Misdiagnosis of Cervical Spondylotic Amyotrophy as a Rotator Cuff Tear*, 12 ASIAN SPINE J. 69 (2018) (Ex. A, Tab 2) (“Iwata”)).

Dr. Leist opined that, based on Petitioner's history of continued shoulder pain, it is likely that he injured his rotator cuff in January or February 2015, and his continued weightlifting after the injury might have exacerbated his right shoulder problems. First Leist Rep. at 6. Dr. Leist explained that there is no known mechanism for the Prevnar 13 vaccine to cause a rotator cuff tear when pain presents 42 days after vaccination, but heavy weightlifting can cause such tears. *Id.*

Alternatively, Dr. Leist opined that if Petitioner's "condition is in fact [PTS] then weight lifting, an activity that he was reportedly engaged in at the time pain and weakness evolved, may be its cause." First Leist Rep. at 5. The Prevnar 13 vaccine is not known to cause PTS, but stressful exercise, such as avid weightlifting, is associated with PTS. *Id.* (citing Feinberg & Radecki).

As for timing, Dr. Leist opined that Petitioner's symptoms arose outside an accepted time interval. First Leist Rep. at 6. Dr. Leist disagreed with Dr. Steinman's attempt to analogize PTS and GBS, commenting that they are "distinct conditions," with PTS "believed to be an axonal process with significant to complete denervation of involved muscles." *Id.* at 5 (citing Feinberg & Radecki). Thus, Dr. Leist did not believe the medically appropriate time interval for GBS following influenza vaccination was applicable to PTS. *Id.* Moreover, the accepted timing for GBS following influenza vaccination is no more than 42 days, but Petitioner's "PTS" symptoms began outside even that interval. *Id.* Finally, the Vaccine Injury Table presumes causation for PTS following tetanus toxoid-containing vaccines no more than 28 days after vaccination. *Id.* Again, Petitioner's symptoms began outside that interval. *Id.*

### 3. Dr. Steinman's Second Expert Report

In his second expert report, Dr. Steinman responded to six questions raised by Special Master Oler in an April 3, 2019 order:

1. Do you attribute Petitioner's neck pain to PTS? Why or why not?
2. You note the onset of Petitioner's PTS at a minimum occurred 41 days after vaccination. Presumably this places onset at January 1, 2015. If onset occurred on January 31, 2015, however, it would be 71 days after vaccination. Do you find a 71-day onset to be an appropriate temporal interval in this case? What about an onset date between 42 and 70 days post vaccination?
3. In your report, you state "[t]he key issue here is timing of the PTS and timing of the rotator cuff tear." ECF No. 26 at 5. Please discuss the significance of the timing of the rotator cuff tear.
4. Do you agree that the MRI performed on September 9, 2015 documented a rotator cuff tear?

5. Do you agree with Dr. Leist that rotator cuff tears can cause atrophy of muscles of the shoulder girdle, including the triceps and/or biceps muscle? Why or why not?

6. Would you expect PTS to be chronic in nature (i.e., lasting for more than one year)? Why or why not?

ECF No. 35.

First, Dr. Steinman opined Petitioner's neck pain was more likely due to PTS than a rotator cuff tear. Second Steinman Rep. at 1 (referencing Ex. 5 at 5). In support of his position, Dr. Steinman referenced a case report of a 44-year-old man who was involved in a rear-end motor vehicle collision, complained of right-sided shoulder pain immediately thereafter, developed neck pain two and a half years later, and ultimately was diagnosed with PTS.<sup>10</sup> *Id.*; see Adam L. Schreiber et al., *Expanding the Differential of Shoulder Pain: Parsonage Turner Syndrome*, 109 J. AM. OSTEOPATH ASSOC. 415 (2009) (Ex. 48) ("Schreiber").

Second, Dr. Steinman opined there was insufficient evidence to discern whether an onset between 42 and 70 days would be medically acceptable. Second Steinman Rep. at 2. He pointed to a reported case of PTS "7 months following a febrile illness." *Id.* (citing A. Michotte et al., *Recurrent forms of sporadic brachial plexus neuropathy*, 90 CLIN. NEUROL. NEUROSURG. 71 (1988) (Ex. 50) ("Michotte")).

Third, Dr. Steinman discussed the significance of the timing of Petitioner's rotator cuff injury. Second Steinman Rep. at 2. He opined that it is unclear when Petitioner's rotator cuff tear occurred, whereas "we know exactly when the immunization occurred." *Id.* He noted that there is no definitive time for onset of Petitioner's PTS, only that onset began sometime in January 2015. *Id.*

Fourth, Dr. Steinman agreed that the MRI performed on September 9, 2015, documented a rotator cuff tear. Second Steinman Rep. at 3.

Fifth, Dr. Steinman did not directly respond to the question of whether a rotator cuff tear can cause atrophy of the muscles of the shoulder girdle. Instead, he commented that "[t]here is abundant literature that PTS can also cause atrophy of the shoulder girdle, including the biceps and triceps muscle." *Id.* (citing Peter Tsairis et al. *Natural history of brachial plexus neuropathy: Report of 99 patients*, 27 ARCH NEUROL 109 (1972) (Ex. 51) ("Tsairis")); Schreiber).

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<sup>10</sup> Dr. Steinman also discussed the difference between cervical radiculopathy ("CR") and PTS. Both conditions are characterized by pain in the cervical spine, shoulder, and upper extremity; however, "[s]ymptoms of CR are exacerbated with neck movements, while symptoms related to PTS should not be exacerbated with neck movements." *Id.* (quoting Christopher J. Mamula et al., *Cervical Radiculopathy or Parsonage-Turner Syndrome: Differential Diagnosis of a Patient With Neck and Upper Extremity Symptoms*, 35 J. OF ORTHOPAEDIC & SPORTS PHYSICAL THERAPY 659 (2005) (Ex. 49) ("Mamula")). Dr. Steinman acknowledged that "Dr. Hardy did not distinguish his preference for PTS" and that the medical records did not report whether Petitioner's symptoms were exacerbated by neck movement. *Id.* at 1-2.

Sixth, Dr. Steinman opined that the “sequela of PTS can make the residual deficits a chronic disorder,” capable of enduring for more than one year. Second Steinman Rep. at 3.

#### 4. Dr. Leist’s Second Expert Report

Responding to Dr. Steinman and to Special Master Oler’s inquiries, Dr. Leist opined that it “is well documented in the literature that the likelihood of clinically silent and apparent rotator cuff injury increases with age.” Second Leist Rep. at 1 (citing Senthil Sambandam et al., *Rotator cuff tear: an evidence based approach*, 6 WORLD J. ORTHOP. 902 (2015) (Ex. C, Tab 3) (“Sambandam”)). A high proportion of rotator cuff tear patients are asymptomatic, making early diagnosis challenging. Second Leist Rep. at 2. “Pain and decreasing shoulder power and function should alert the heedful practitioner in recognizing promptly the onset or aggravation of existing [rotator cuff] tears.” *Id.* (quoting Sambandam at 902). Petitioner was in his late sixties at the time of vaccination, and he had shoulder pain, weakness, and progressive atrophy in the months before he was seen by Drs. Dageforde and Hardy, indicating a possible rotator cuff tear. *Id.* at 1-2. Nonetheless, Drs. Dageforde and Hardy did not consider a rotator cuff tear and did not order tests relating to a possible tear. *Id.* (citing Ex. 5 at 5; Ex. 7 at 8). When Petitioner had an MRI in September 2015 after seeing Dr. Walker, he was found to have chronic changes and rotator cuff tears in several locations. *Id.* (citing Ex. 9 at 1, 6).

Dr. Leist reiterated that the fact that Petitioner reported six months of right shoulder pain at his August 12, 2015 visit with Dr. Walker was not consistent with PTS, but it was consistent with a rotator cuff injury. Second Leist Rep. at 2 (citing Ex. 9 at 1). Petitioner’s rotator cuff injury “more likely than not preceded” the subject vaccination and became symptomatic in January 2015 “through progression of structural changes possibly furthered by ‘avid weight lifting.’” *Id.* at 2-3 (citing Ex. 5 at 5-6).

Dr. Leist explained that muscle atrophy can occur with rotator cuff injuries and tears. Second Leist Rep. at 4; Sambandam. Rotator cuff tears can affect the long head of the triceps. *Id.* (citing Hisayo Nasu et al., *Anatomic study on the origin of the longhead of the triceps brachii*, 3 JSES OPEN ACCESS 5 (2019) (Ex. C, Tab 2) (“Nasu”)). Furthermore, full-thickness rotator cuff tears “develop distinct chronic pathological changes due to muscle retraction, fatty infiltration and muscle atrophy.” *Id.* at 5; *see also* Sambandam. By contrast, functional recovery following PTS is good in most cases, and one of the largest natural history studies of PTS found that 89% of patients had full functional recovery at 3 years. *Id.*; *see* Tsairis.

Dr. Leist noted that Dr. Steinman submitted Michotte as a case report of PTS occurring “7 months following a febrile illness.” Second Leist Rep. at 3. But the patients in that report did not receive any vaccine, including Prevnar 13. *Id.* Moreover, the second patient in Michotte had a very different clinical course than Petitioner’s and did not develop PTS seven months after an illness, as Dr. Steinman claimed. *Id.*

Dr. Leist again concluded that Petitioner’s symptoms that began “in January 2015 were more likely due to complex injury of right shoulder structures preceding the vaccination” and were exacerbated by his age and workout routine, not his vaccination. Second Leist Rep. at 5.

## 5. Emanual Maverakis, M.D.: First Expert Report

Dr. Maverakis submitted two reports in this case and testified at the entitlement hearing. Ex. D (“First Maverakis Rep.”); Ex G (“Second Maverakis Rep.”); Tr. at 233-86.

Dr. Maverakis earned his M.D. from Harvard Medical School in 2003 and is board certified in clinical informatics and dermatology. Ex. E (“Maverakis CV”) at 2; Tr. at 234. He is a member of the American Association of Immunologists and primarily researches immune-mediated diseases. Maverakis at 31; Tr. at 235. He is a professor at the University of California, Davis (“UC Davis”) in the Departments of Dermatology and Medical Microbiology and Immunology; he is also the Director of Immune Monitoring Shared Resource and Associate Director of Metabolism and Immunologic Health at UC Davis. Maverakis CV at 3; Tr. at 236. Additionally, he is an elected fellow of the American Society of Clinical Investigation and the California Academy of Sciences. Maverakis CV at 3; Tr. at 235-36. He sees roughly 40 patients per week for skin-related immune-mediated diseases. *Id.* at 237.

Dr. Maverakis opined that Petitioner’s PTS diagnosis, and the conclusion that it was caused by the subject vaccination, are “not supported by the totality of evidence in [Petitioner’s] medical record.” First Maverakis Rep. at 8. Citing a number of papers, Dr. Maverakis noted that “[PTS] is a rare syndrome of unknown etiology that is most commonly associated with recent history of immunization, surgery, trauma, or infection. It also sometimes presents in the setting of autoimmunity.” *Id.* However, although PTS has been temporally associated with vaccination, Dr. Maverakis could not find any literature reporting cases of PTS following Prevnar 13 vaccination. *Id.*

Although Dr. Maverakis deferred to Dr. Leist regarding Petitioner’s diagnosis, he had several comments: first, Petitioner had a long history of sensorimotor polyneuropathy, which preferentially affected his lower extremities, but with “documented upper extremity involvement at least as far back as 2010.” First Maverakis Rep. at 8. Second, his MRI revealed a full-thickness rotator cuff tear, as well as AC arthropathy and external impingement. *Id.* Third, PTS often resolves spontaneously, with 75% of patients recovering in 2 years and 90% recovering in 3 years. *Id.* By contrast, it is extremely common for patients with chronic rotator cuff injuries to have chronic symptoms. *Id.* Here, although Petitioner experienced some degree of improvement, his chronic symptoms did not match the typical longitudinal course of PTS. *Id.* Fourth, if Petitioner’s diagnosis was, in fact, PTS, then it might have been caused by the trauma of the full-thickness rotator cuff tear in his right shoulder, as trauma is a known cause of PTS. *Id.*

Dr. Maverakis disagreed with Dr. Steinman’s opinion that the definition of PTS “perfectly fits Petitioner’s case.” First Maverakis Rep. at 8 (citing Ex. 12 at 5). Instead, Petitioner clearly “had signs and symptoms of a rotator cuff tear” when he presented to Dr. Hardy. *Id.* at 8-9. He also had several risk factors that should have prompted Dr. Hardy to include a rotator cuff injury as part of his differential. *Id.* at 9. For example, rotator cuff tears are common for Petitioner’s age group and are associated with weightlifting. *Id.* Dr. Hardy, though, did not order any imaging of the rotator cuff or examine it. *Id.* Later, Dr. Walker appropriately suspected that Petitioner had a rotator cuff tear, examined his rotator cuff, ordered an MRI, and confirmed the diagnosis. *Id.*

Dr. Maverakis also disputed Dr. Steinman's causation theory. He commented that the two studies Dr. Steinman relied on were from the 1970s, and neither was relevant to this case. First Maverakis Rep. at 9. In one study, "polyganglionic radiculoneuritis was induced in rhesus monkeys by injection with rabbit sciatic nerve myelin," and in the other, "guinea pigs were sensitized with peripheral nerve myelin." *Id.* (citing Wisniewski and Brostoff).

Dr. Maverakis also noted that Dr. Steinman supported his theory by "describing how a variety of autoimmune neurological diseases" can have autoantibodies to phosphatidylcholine structures, which are present in Prevnar 13. First Maverakis Rep. at 9. But the diseases cited by Dr. Steinman, GBS and multiple sclerosis, are systemic autoimmune diseases, whereas Petitioner's injury was localized to his right side. *Id.* Dr. Maverakis questioned why an immune response would stay so localized. *Id.*

#### 6. Dr. Steinman's Third Expert Report

Dr. Steinman remarked that if he saw Petitioner in his neurology clinic, he would seek a consult from an orthopedic surgeon regarding Petitioner's rotator cuff injury. Third Steinman Rep. at 1. However, in his view, Petitioner's rotator cuff injury became symptomatic due to his PTS, which in turn was caused by the subject vaccination. *Id.* He disagreed with Dr. Leist's opinion that Petitioner's symptoms were the result of a preexisting right shoulder injury. *Id.*

Lastly, Dr. Steinman opined that a systemic autoimmune disease, including one directed against phosphatidylcholine structures, can and often will have localized symptoms. Third Steinman Rep. at 2. Thus, it was appropriate to analogize Petitioner's PTS to conditions like GBS and multiple sclerosis. *Id.*

#### 7. Daniel Carr, M.D.: Expert Report

Dr. Carr submitted one report in this case. Ex. 62 ("Carr Rep."). He also testified at the entitlement hearing. Tr. at 117-41.

Dr. Carr earned his M.D. from the University of Vermont in 1980. Ex. 63 ("Carr CV") at 1; Tr. at 118. Although he is currently retired from active surgical practice, he was an orthopedic surgeon for 30 years and still maintains his medical license and is board certified. Carr CV at 1; Tr. at 117; Carr Rep. at 2. He has an extensive history in sports medicine: he treated Olympic athletes for 12 years, including treating weightlifters at the 1996 Olympic games in Atlanta and serving as the head U.S. team physician at the 2002 Olympic games in Salt Lake City. Carr CV at 1-2; Tr. at 118. Additionally, he has provided orthopedic consultations for professional sports teams, such as the Chicago Cubs, and he was an orthopedic surgeon for the College of William and Mary and Christopher Newport University. Carr CV at 2; Tr. at 118. Currently, Dr. Carr cares for orthopedic patients at his local free medical clinic, is a volunteer surgeon in St. Vincent and the Grenadines, and serves on multiple medical advisory boards. Carr CV at 2; Carr Rep. at 2.

Dr. Carr deferred to Dr. Steinman on the question of whether the Prevnar 13 vaccine can cause PTS. Carr Rep. at 6. He opined that Petitioner's muscle atrophy, wasting, and secondary

asymmetric upper extremity weakness from PTS caused Petitioner's "undiagnosed and asymptomatic rotator cuff tear to become symptomatic and require treatment." *Id.* He explained that more than 50% of patients over the age of 60 have rotator cuff pathologies, ranging from tendinitis to complete tears, the majority of which are asymptomatic. *Id.* Moreover, ardent weightlifters have a higher incidence of rotator cuff tears than the general population due to the vigorous strain they continuously place on their shoulders. *Id.* at 6-7. As a result, "most orthopedic physicians are reluctant to order MRIs on the shoulders of minimally symptomatic weightlifters," because they know that they will most likely find considerable pathology from years of significant overuse. *Id.* at 7. He explained:

Athletes, particularly weightlifters, but also swimmers and overhead throwing athletes, will frequently develop symptomatic shoulder pathology when their strength and flexibility become unbalanced. This is a result of their posterior musculature being stronger than their anterior musculature or vice versa. This imbalance causes the shoulder joint to produce an abnormal shearing force creating a poorly controlled and destructive shifting forward and back during use. This abnormal biomechanical force incites inflammation, swelling and pain. Additional tearing of the rotator cuff, labrum or degenerative joint disease can and do occur if the disease and imbalance persist or is not corrected.

*Id.* According to Dr. Carr, Petitioner had this "precise injury pattern" following the subject vaccination. *Id.*

Dr. Carr further explained that it is unlikely that the subject vaccination directly caused Petitioner's rotator cuff tear; rather, it is more likely that the tear was present, relatively small, and centrally located prior to vaccination. Carr Rep. at 7. Because Petitioner maintained his strength and balance through weightlifting, neither he nor anyone else would suspect that a problem existed in his right shoulder; however, PTS and its accompanying symptoms created imbalance in Petitioner's shoulder, putting more stress on his rotator cuff, and causing his subsequent symptoms. *Id.* Dr. Carr opined that had it not been for PTS, it is highly unlikely that Petitioner would have had any symptoms or even known that he had a rotator cuff tear. *Id.* at 8. Dr. Carr predicted that an MRI of Petitioner's left shoulder "would very likely show pathology and perhaps even a rotator cuff tear," but he remains asymptomatic on his left side because he has not had PTS on that side and therefore "has a strong balanced supporting musculature and strength." *Id.*

#### 8. Dr. Leist's Third Expert Report

Dr. Leist commented that weightlifters, such as Petitioner, have a greater risk of symptomatic rotator cuff pathology due to the strain on the shoulder girdle, and the record here shows Petitioner "continued to lift weights even after onset and during progression of symptoms." Third Leist Rep. at 2. Furthermore, "pain associated with [PTS] is almost always self-limiting and normally lasts 1 to 2 weeks," whereas Petitioner's shoulder pain was chronic and "exacerbated by movement against resistance and improved with icing and rest." *Id.* Dr. Leist reiterated that

“aggravation of pain with movements and onset more than 42 days after vaccination is not consistent with [PTS].” *Id.*

In PTS, it is likely that “some degree of improvement would be observed following the maximal clinical presentation,” but in Petitioner’s case there was no improvement and his shoulder pain persisted. Third Leist Rep. at 2. Dr. Leist opined that Petitioner’s clinical course and symptoms were not typical for PTS. *Id.*

Lastly, Dr. Leist challenged Dr. Carr’s underlying assumption that Petitioner suffered from PTS and that the subject vaccination caused his PTS. Third Leist Rep. at 2. “Dr. Carr and Dr. Steinman do not provide any evidence that would indicate that [Petitioner] specifically suffered from [PTS] and not a symptomatic rotator cuff injury[.]” *Id.* Moreover, by relying on Dr. Steinman’s opinion on causation, Dr. Carr did not address whether the subject vaccination can cause PTS, consider the time interval for onset, or demonstrate that it is more likely Petitioner suffered from PTS and not an exercise-associated shoulder injury. *Id.* at 3. Dr. Leist opined that Drs. Steinman and Carr based their opinions on “temporality” despite Petitioner’s symptoms arising 42 days or more after vaccination. *Id.*

#### 9. Dr. Maverakis’ Second Expert Report

Dr. Maverakis maintained that Petitioner did not have PTS. Second Maverakis Rep. at 1. Petitioner was 67 at the time of vaccination, but PTS is increasingly rare in patients over 60. *Id.* Also, the only physician to diagnose PTS, Dr. Hardy, did not order imaging to confirm the diagnosis or rule out other potential diagnoses, even though Petitioner’s exam and clinical course were not consistent with a classic case of PTS. *Id.* at 1-2. When Petitioner eventually did get an MRI, it did not show focal intrinsic nerve constrictions, which are seen in the vast majority of PTS patients, but it did show a rotator cuff tear. *Id.* at 1.

By contrast, rotator cuff injuries are very common, and the risk increases with age and with activities like weightlifting. Second Maverakis Rep. at 1. Notably, orthopedist Dr. Walker found Petitioner’s presentation was more consistent with a rotator cuff tear than PTS. *Id.* at 1-2. Similarly, the second orthopedist, Dr. Higgs, did not “state any action to take with regards to [PTS].” *Id.* at 4. Dr. Maverakis also pointed out that the atrophy observed in Petitioner’s right triceps and pectoral muscles could have been caused by his rotator cuff tear, which would have impeded his ability to lift weights on that side. *Id.* at 3.

Dr. Maverakis disagreed with Dr. Carr’s opinion that Petitioner’s rotator cuff would not have become symptomatic but-for PTS caused by vaccination. The medical literature indicates that “asymptomatic rotator cuff tears are likely to become symptomatic over time.” Second Maverakis Rep. at 4.

On causation, Dr. Maverakis questioned the references cited by Dr. Steinman, observing that they were “nearly 50 years old and [had] nothing to do with brachial neuritis presenting after vaccination.” Second Maverakis Rep. at 5. He pointed out that there are “no reports of patients with [PTS] having phosphatidyl choline antibodies” and there also are “no reports linking IL-1 or IL-18 to [PTS].” *Id.* at 5-6. IL-1 and IL-18 are innate cytokines that are unlikely to induce

autoimmunity. *Id.* at 6. Also, the studies Dr. Steinman cited showed a lipid-specific immune response in MS but did not demonstrate that lipids can induce autoimmunity. *Id.* at 7. Dr. Maverakis noted that the majority of molecular mimicry studies “are based on *in vitro* cross-reactive immune response to self and foreign antigens,” but demonstrating such cross-reactivity “does not necessarily mean that priming an individual with the mimic will induce autoimmunity.” *Id.*

Finally, Dr. Maverakis disagreed with Dr. Steinman that PTS can be analogized to GBS, as the diseases have “strikingly different clinical presentations.” Second Maverakis Rep. at 7. Further, IL-1 and IL-18 have not been shown to be the major cytokines in GBS and also have not been associated with PTS. *Id.*

#### 10. Geoffrey Abrams, M.D.: Expert Report

Dr. Abrams submitted one report in this case and testified at the entitlement hearing. Ex. H (“Abrams Rep.”); Tr. at 181-233.

Dr. Abrams earned his M.D. from the University of California, San Diego in 2007 and is board certified in general orthopedic surgery with a subspecialty certificate in orthopedic sports medicine. Ex. I (“Abrams CV”) at 1-2; Tr. at 181-82. He is an associate professor of orthopedic surgery at Stanford University School of Medicine, where he is also the director of the Lacob Family Sports Medicine Center for varsity athletes at Stanford. Abrams CV at 1; Tr. at 182. Additionally, Dr. Abrams is the team physician for the San Francisco 49ers and Golden State Warriors. Abrams CV at 24; Tr. at 182. He is a reviewer for several journals, including principal reviewer for the *American Journal of Sports Medicine*, and he has published more than 80 peer-reviewed articles. Abrams CV at 10-19; 23. Although he is not an expert in PTS, cases of PTS come up in the general course of his orthopedic practice; he sees “anywhere from zero to five patients a year with PTS.” Tr. at 183. Dr. Abrams refers patients with suspected PTS to a specialist. *Id.*

Dr. Abrams noted that he and Dr. Carr should be able to agree that Petitioner’s rotator cuff pathology was present prior to the subject vaccination. Abrams Rep. at 4. They also should agree that “that muscular imbalances and weakness around the shoulder girdle can lead to shoulder pain, and in some cases may be associated with previously asymptomatic rotator cuff pathology becoming symptomatic.” *Id.* But Dr. Abrams noted that the pectoral and triceps muscles, where Petitioner had atrophy, are not known to be associated with this phenomenon. *Id.* He stated that it “would be very unlikely for weakness of the triceps and/or pectoral muscles” to cause a preexisting rotator cuff tear to become symptomatic; it is “much more likely” that Petitioner’s preexisting rotator cuff injury became symptomatic “through continued exercise and weight lifting.” *Id.* at 6 (citing Morey J. Kolber et al., *Shoulder Joint and Muscle Characteristics in the Recreational Weight Training Population*, 23 THE J. OF STRENGTH & CONDITIONING RSCH. 148 (2009) (Ex. H, Tab 9) (“Kolber”); Thomas J. Neviaser, *Weight Lifting Risks and Injuries to the Shoulder*, 10 CLINICS IN SPORTS MED. 615 (1993) (Ex. H, Tab 10) (“Neviaser”).

Dr. Abrams did not believe that Dr. Carr’s discussion of how overhead-throwing athletes and swimmers develop symptomatic shoulder pathology when their shoulders become unbalanced

was applicable to Petitioner. Abrams Rep. at 4. He explained that “[i]n overhead athletes, which are almost all significantly younger in age than petitioner, it is almost unheard of to have full thickness rotator cuff tears as well as arthritis.” *Id.* The imbalance they experience “stems from repetitive loading of the shoulder capsular tissue during the follow through motion, causing tightness in this tissue, and therefore altering the biomechanics and range of motion (particularly loss of internal rotation) of the shoulder.” *Id.* Similarly, “instability (imbalance) secondary to ligamentous laxity” as a cause of “swimmer’s shoulder” is not relevant, as that usually affects young females who are double jointed. *Id.* at 5. Pectoral and/or triceps muscle weakness is not a cause of “swimmer’s shoulder.” *Id.*

Dr. Abrams also pointed out that there is “a somewhat analogous clinical situation to [P]etitioner’s condition for his pectoral muscle weakness and atrophy noted on exam – a chronic pectoralis tendon rupture.” Abrams Rep. at 6. This condition involves pectoral atrophy but is not associated with shoulder pain, suggesting atrophy of the pectoral muscles does not cause such pain. *Id.* He added:

The pectoralis major is responsible for internal rotation and adduction of the humerus, yet the orthopedic surgeon examining petitioner in January 2016 clearly records weakness with external rotation – consistent with rotator cuff pathology as the rotator cuff muscles are the only external rotators of the shoulder. This visit also documents triceps atrophy, but not pectoral atrophy. Given its anatomical location, the triceps muscle is not thought to contribute to glenohumeral mechanics and therefore is not known to be a clinical factor in the development of symptomatic shoulder pathology.

*Id.* (emphasis in original and citations omitted).

Dr. Abrams noted that the remaining question was what might have caused a previously asymptomatic rotator cuff tear to become symptomatic in Petitioner. Abrams Rep. at 6. He pointed out that the literature shows periscapular weakness is associated with such symptoms. *Id.* (citing Anthony M. Barcia & Justin L. Makovicka, *Scapular Motion in the Presence of Rotator Cuff Tears: A Systemic Review*, J. OF SHOULDER & ELBOW SURGERY (Journal Pre-Proof) (2021) (Ex. H, Tab 12) (“Barcia & Makovicka”); Hiroaki Ishikawa et al., *Differences in scapular motion and parascapular muscle activities among patients with symptomatic and asymptomatic rotator cuff tears, and healthy individuals*, 5 JSES INT’L 238 (2021) (Ex. H, Tab 13) (“Ishikawa”); Xavier Robert-Lachaine et al., *Scapulohumeral rhythm relative to active range of motion in patients with symptomatic rotator cuff tears*, 25 J. OF SHOULDER & ELBOW SURGERY 1616 (2016) (Ex. H, Tab 14) (“Robert-Lachaine”)). This type of weakness is not known to be significantly affected by either the pectoral muscle or the triceps. *Id.* at 6-7.

Petitioner had several risk factors associated with symptomatic rotator cuff tears, including a positive impingement sign, weakness in external rotation, and weakness in his dominant arm. Abrams Rep. at 7. Dr. Abrams concluded that it is “overwhelmingly probable” that Petitioner’s “shoulder pain arose from the natural course of his pre-existing shoulder condition rather than

some unknown and undescribed mechanism of imbalance in the shoulder stemming from his pectoral and triceps muscle atrophy.” *Id.*

Additionally, Dr. Abrams opined that Petitioner “does not fit the typical PTS clinical course or symptomatology.” Abrams Rep. at 7. He explained:

From an orthopedic perspective, PTS typically presents as upper extremity pain followed by muscular weakness and/or atrophy. It has been noted that 90% of PTS cases present with pain as the first symptom with weakness noted in the first two weeks in a great majority of cases. Sensory disturbances are also seen in about 80% of cases. Furthermore, while the triceps muscle (radial nerve) can be [a]ffected, it is extremely uncommon for the pectoralis muscle to be involved in PTS (medial and lateral pectoral nerves), and even less common to see a combination of pectoral and triceps involvement.

*Id.* at 8 (citations omitted). During Petitioner’s first visit with Dr. Dageforde, he reported atrophy, but no pain; then, during his visit with Dr. Hardy in May 2015, he again reported atrophy and that he developed neck discomfort in January 2015. *Id.* He did not report shoulder pain until his visit with Dr. Walker in August 2015, at which time he reported six months of shoulder pain. *Id.* Based on these reports, Dr. Abrams placed onset for neck pain in January 2015 and shoulder pain in February 2015, more than two and three months after vaccination. *Id.* Dr. Abrams opined that “this clinical time course does not fit with the most common clinical presentation of PTS documented in larger studies of patients with PTS.” *Id.* Further, Petitioner had “a history of EMG-diagnosed sensorimotor polyneuropathy involving upper and lower extremities years before his vaccination which may place him at risk for developing future peripheral nerve disorders.” *Id.* (citing Ex. 5 at 1).

## B. Expert Testimony

### 1. Dr. Steinman’s Testimony

Dr. Steinman was qualified as an expert in neurology and neuroimmunology. Tr. at 74. He testified that the cause of PTS “is not known in a decisive way,” but vaccination, infection, and trauma are triggers of the condition. *Id.* at 78.

With respect to the mechanism of causation, Dr. Steinman explained that inflammatory neuropathies such as GBS involve an immune response to phospholipids present in the myelin sheath of nerves. Tr. at 79. “So the general idea is that there’s a component of the vaccine that is a molecular mimic . . . [that] has structural similarity or identity to components of the myelin sheath.” *Id.* Additionally, the Prevnar 13 vaccine has an adjuvant, alum, that elicits production of two cytokines, IL-1 and IL-18, which are elevated in inflammatory neuropathies like GBS. *Id.* at 80-81.

Elaborating on his molecular mimicry theory, Dr. Steinman explained that, based on the information he was able to gather, the Prevnar 13 vaccine contains a “building block,” phosphatidylcholine, which is identical to a component of the myelin sheath. Tr. at 81-82. This component provokes an immune response to the myelin. *Id.* at 83. With respect to the alum adjuvant theory, Dr. Steinman testified that alum “has been well studied” in relation to GBS. *Id.* at 83-84. He has done research on animal models of Experimental Allergic Neuritis (“EAN”), a GBS-like condition. *Id.* at 84. Giving EAN models antibodies to IL-18 produces improvements in paralysis, suggesting that elevated IL-18 is associated with the pathology of EAN and GBS. *Id.* GBS and PTS are analogous, and thus a similar mechanism of causation could apply to PTS. *Id.* at 84-85. On cross, he acknowledged that he used Complete Freund’s Adjuvant (“CFA”) in his animal studies, which is very toxic and not used in humans. *Id.* at 99.

Dr. Steinman’s diagnostic opinion relied heavily on the treating physicians who examined and diagnosed Petitioner. Tr. at 86-87. On cross, though, he acknowledged that Petitioner was not given an EMG to confirm PTS, despite his preexisting history of neck pain and polyneuropathy of both the upper and lower extremities. *Id.* at 95. He opined that, in addition to PTS, Petitioner had a rotator cuff injury. *Id.* at 86. He agreed that Petitioner’s bodybuilding could have contributed to the rotator cuff injury, but he did not believe bodybuilding could have caused his PTS. *Id.* at 86, 91. Although strenuous exercise can trigger PTS, that was unlikely here because Petitioner did not have PTS until after the vaccination, despite being a lifelong weightlifter. *Id.* at 98.

With respect to onset, Dr. Steinman pointed to Petitioner’s testimony that his symptoms began in late December 2014, rather than in January 2015, as Dr. Steinman recounted in his reports. Tr. at 88. However, even if Petitioner’s neck pain did not start until January, about 41 days post-vaccination, “the time frame is still permissible for a diagnosis of [PTS].” *Id.* He opined that this timeframe would be medically acceptable based on the latency period associated with GBS following flu vaccination. *Id.* at 89. He further testified that, based on the literature, an onset timeframe of up to 10 weeks would be acceptable. *Id.* at 109.

On cross, Dr. Steinman admitted that he did not have any literature describing PTS occurring after Prevnar 13 vaccination. Tr. at 95-96. The literature he cited did not find anti-phospholipid antibodies in PTS patients; nor was there any evidence Petitioner had such antibodies. *Id.* at 101. There also was no evidence in the medical records that Petitioner had elevated levels of IL-1 or IL-18. *Id.* at 107.

## 2. Dr. Carr’s Testimony

Dr. Carr was qualified as an expert in orthopedic surgery and sports medicine. Tr. at 120. He testified that Petitioner’s past as a weightlifter and steroid user likely would have caused him to develop asymptomatic rotator cuff tears. *Id.* at 120-21. Due to PTS, he developed significant muscle atrophy in his right pectoralis major and triceps. *Id.* at 121-22. This atrophy, combined with continued weightlifting, caused his shoulder to become “unbalanced,” which in turn caused the rotator cuff tear to become symptomatic. *Id.* at 122-23. Dr. Carr did not feel Petitioner’s past steroid use, which ceased more than 20 years earlier, contributed to his condition. *Id.* at 123-24.

Dr. Carr was unable to precisely date when Petitioner's rotator cuff tear occurred, noting Petitioner's testimony that he had intermittently experienced rotator cuff problems for years prior to the vaccination. Tr. at 125. There was, however, a high degree of probability that, at the time Petitioner was diagnosed by Dr. Hardy with PTS in May 2015, he had a preexisting tear. *Id.* at 125-26. By the time Petitioner saw Dr. Walker in August 2015, the tear had become symptomatic. *Id.* Dr. Carr explained how that might have occurred:

So once a perfectly balanced shoulder gets out of sync, that is the front can't pull or do its fair share compared to the back, the stress shifts, and that puts more pressure on the arthritic joint. It causes further irritation and pain within the rotator cuff itself and it sets up this cascade effect that all of a sudden something that functioned normally and no one would have any idea this gentleman with these huge arms and shoulders would ever have a rotator cuff tear, all of a sudden become symptomatic.

*Id.* at 128. In other words, after Petitioner experienced muscle wasting due to PTS, he continued to exercise, putting more strain on his shoulder, which "threw his shoulder off, causing a shearing effect and therefore, pain in his shoulder, which caused him to seek medical advice and an orthopedic surgeon." *Id.* at 129. Dr. Carr opined that, but for the PTS Petitioner developed as a result of his vaccination, his rotator cuff tear would likely have remained asymptomatic. *Id.*

Dr. Carr did not believe Petitioner's weightlifting could have caused him to develop PTS. Tr. at 131. He had been weightlifting for many years without incident and did not do "anything out of the ordinary" that could have triggered the condition. *Id.*

On cross, Dr. Carr acknowledged that he could find no literature showing how a rotator cuff injury would be affected by pectoralis major and triceps atrophy, as seen in Petitioner. Tr. at 134. He further agreed that different muscles are used by overhead throwing athletes than those affected in Petitioner's case. *Id.* He noted, however, that Petitioner had scapular dyskinesis, which is seen in swimmers and overhead throwers. *Id.* at 135. He acknowledged that Petitioner had arthritis in the right shoulder before his vaccination, which also causes shoulder pain. *Id.* at 136. Finally, he concurred that most asymptomatic rotator cuff tears do eventually become symptomatic, and that risk increases with age. *Id.* at 136-37.

In response to questions from Special Master Oler, Dr. Carr testified that it was more likely Petitioner had an asymptomatic rotator cuff tear before the vaccination, which later became painful, than that he developed a new, symptomatic tear after vaccination. Tr. at 137. This is because the MRI showed many changes consistent with a longstanding condition, as is frequently seen in weightlifters. *Id.* at 137-38. He also pointed to the fact that the onset of Petitioner's weakness was gradual, not sudden, indicating that it was due to PTS and not a major muscle injury. *Id.* at 140.

### 3. Dr. Leist's Testimony

Dr. Leist was qualified as an expert in neurology and neuroimmunology. Tr. at 146. He disagreed that Petitioner had PTS, as the typical clinical course for the disease involves an insidious onset of “searing” and unrelenting pain. *Id.* at 147-48. Generally, the pain subsides after about two weeks, and weakness develops. *Id.* at 148. Most patients then have “some degree of recovery.” *Id.* at 149. Petitioner did not have the searing pain associated with PTS. *Id.* at 158.

Dr. Leist disagreed with Dr. Steinman that it was unnecessary to confirm Petitioner’s PTS diagnosis with an EMG. Tr. at 155. PTS is much rarer than other conditions that might mimic it, such as rotator cuff tears and adhesive capsulitis. *Id.*

Also, Petitioner had a “known history of cervical problems” that predated the vaccination. Tr. at 149-50. In 2010, he was observed to have balance problems, peripheral neuropathy, and decreased ROM of the neck and back. *Id.* at 150-51. In 2018, an MRI of the cervical spine showed chronic changes in the cervical spine. *Id.* at 151. Since Dr. Leist’s last report was filed, Petitioner had further worsening of this disease. *Id.* at 159. This was more likely than PTS to have caused the “thenar atrophy” seen between Petitioner’s thumb and forefinger. *Id.* at 151-52. Dr. Leist also pointed out that Petitioner also had biceps atrophy at the time of his initial post-vaccination exam, which is correlated with cervical disc degenerative disease. *Id.* at 153. On cross, however, Dr. Leist acknowledged that Dr. Dageforde did not document biceps atrophy during his initial examination of Petitioner, though Petitioner did report it. *Id.* at 168-69. He further admitted that Petitioner had several exams in 2015 in which he had full ROM of the cervical spine. *Id.* at 170, 172.

Dr. Leist opined that, even if Petitioner had PTS, it was more likely caused by vigorous weightlifting, which is a recognized cause of PTS, than by the subject vaccination. Tr. at 158. He did not believe the vaccination played any role in causing Petitioner’s preexisting rotator cuff tear to become symptomatic or in causing his current symptoms. *Id.* at 157-60. A large rotator cuff tear like Petitioner’s would become symptomatic on its own, particularly “in [the] context of continued exercise.” *Id.* at 157. Petitioner’s current condition is “completely independent of any ill effects of the vaccine.” *Id.* at 160.

With respect to onset, Dr. Leist opined that the earliest Petitioner experienced symptoms was January 2015. Tr. at 160. His shoulder pain was “really reported during the summer months, after May of 2015.” *Id.* Thus, even assuming the 3-42-day interval for onset of GBS following flu vaccination applied here, the onset of Petitioner’s symptoms after his Prevnar 13 vaccination fell outside that interval. *Id.* at 161. However, it was inappropriate to use the flu/GBS onset interval here, because GBS involves multiple nerve roots, unlike PTS, and because the pathogenesis of PTS is poorly understood. *Id.* at 162-63. Similarly, Petitioner’s onset fell outside the accepted interval for brachial neuritis/PTS following tetanus vaccination. *Id.* at 161-62.

#### 4. Dr. Abrams's Testimony

Dr. Abrams was recognized as an expert in orthopedic surgery and sports medicine. Tr. at 186. He, too, disagreed with the diagnosis of PTS in Petitioner. *Id.* at 193. First, Petitioner's presenting complaint was not consistent with a "classic" PTS diagnosis, because he complained of atrophy, not shoulder pain. *Id.* Dr. Abrams's PTS patients have "exclusively" presented with fairly severe shoulder pain as their primary complaint. *Id.* at 193-94. On cross, he acknowledged that it is possible for a patient with PTS to present with a different primary complaint. *Id.* at 216.

Second, Petitioner's pattern of atrophy did not match classic PTS. Tr. at 194. The pectoralis muscle is "one of the least affected muscles" in PTS and is "extremely unlikely" to be affected in combination with triceps muscle atrophy. *Id.* at 194-95. Dr. Abrams used a diagram of the brachial plexus to illustrate that the nerves involved in the large majority of PTS cases are different from the nerves for the pectoralis or triceps muscles, making it unlikely that atrophy of the pectoralis and/or triceps is related to PTS. *Id.* at 217-18; *see Ex. L.*

Dr. Abrams opined that Petitioner had a "classic rotator cuff arthropathy," including fairly significant rotator cuff tearing and arthritis. Tr. at 196. The MRI showed several signs consistent with such a tear, including superior migration of the humeral head and bone spurring. *Id.* at 196-97.

Rotator cuff arthropathies are very common. Tr. at 199. Full-thickness rotator cuff tearing occurs in about 50% of patients over 60 years of age. *Id.* at 198. A rotator cuff tear can be caused in part by a "mechanical phenomenon of the tendon rubbing on the bone," but inflammation also plays a role, by leading to collagen breakdown in the tendon. *Id.* at 197. Weightlifting is a risk factor for this condition, as is advanced age. *Id.* at 198-99.

Rotator cuff arthropathies often have an idiopathic onset, with the patient not knowing why their symptoms began. Tr. at 199-200. Typically, tendon tears enlarge over time, worsening arthritis, leading to an "increased clinical symptomology pattern as the months and years go on." *Id.* at 200.

Dr. Abrams disagreed with Dr. Carr's theory that atrophy of Petitioner's pectoralis and triceps muscles (caused by PTS) created an imbalance in Petitioner's shoulder, causing his previously asymptomatic rotator cuff tear to become symptomatic. Tr. at 200. There is no evidence that those two muscles contribute to the balance of the shoulder. *Id.* at 201. To the contrary, data point to other muscles as contributory. *Id.* For example, problems with the periscapular muscles can cause rotator cuff tears to become symptomatic. *Id.* In January 2015, Petitioner was observed to have scapular dyskinesis, in which the scapula is in an abnormal position due to dysfunction of the periscapular muscles. *Id.* at 202-03.

Dr. Abrams distinguished glenohumeral internal rotation deficit ("GIRD"), which Dr. Carr described in his report. Tr. at 203. GIRD typically affects overhead-throwing athletes and involves a posterior tightening of the shoulder capsule. *Id.* at 205. That tightening precludes the arm from moving in internal rotation. *Id.* The mechanics of GIRD "argue[] against the fact that the

pectoralis and triceps are responsible” for shoulder imbalance and consequent rotator cuff symptoms. *Id.* at 205-06. In fact, GIRD leads to scapular dyskinesis. *Id.* at 206. Similarly, “swimmer’s shoulder,” referenced by Dr. Carr, is seen in athletes with excessive mobility of the shoulder. *Id.* at 206-07. That condition also does not involve dysfunction of the pectoralis or triceps muscles. *Id.* at 207.

Dr. Abrams further explained that he frequently sees patients with chronic pectoralis tendon rupture, which occurs where the pectoralis tendon attaches to the humerus. Tr. at 208. Such a rupture can cause atrophy of the pectoralis muscle. *Id.* Generally, these patients do not complain of shoulder pain, even though many of them likely also have rotator cuff pathology due to lifestyle factors such as weightlifting. *Id.* at 209. This provides further evidence that weakness of the pectoral muscle does not cause rotator cuff tears to become symptomatic. *Id.* at 209-10.

Dr. Abrams opined that, according to the medical records, Petitioner’s rotator cuff tear became symptomatic in August 2015, when he complained of right shoulder pain. Tr. at 210. As to the cause of that pain, Petitioner’s continued weightlifting in the months after the vaccination was a “textbook setup of shoulder pain from rotator cuff tear arthropathy.” *Id.* at 211. Dr. Abrams did not believe that, if Petitioner did have PTS, it was caused by the Prevnar 13 vaccine; nor did he believe the vaccine played any role in causing his rotator cuff tear to become symptomatic. *Id.* at 212.

##### 5. Dr. Maverakis’s Testimony

Dr. Maverakis was qualified as an expert in immunology. Tr. at 238. He testified that he had found no literature linking the Prevnar 13 vaccine to PTS. *Id.* at 239. He opined that the causation theories presented by Petitioner’s immunology expert, Dr. Steinman, were not reliable. *Id.* He did acknowledge that the predominant belief is that PTS is an immune-mediated disease. *Id.* at 262.

Addressing the proposed molecular mimicry theory involving antiphospholipid antibodies, Dr. Maverakis testified that “antibodies to lipids are common in the general population, and although they have been linked to autoimmunity, they are not specific for autoimmunity by any means, and there’s no evidence that lipids can induce autoimmunity on their own.” Tr. at 241. The studies cited by Dr. Steinman found anti-lipid antibodies in controls as well as test animals. *Id.* at 242-43. Dr. Maverakis explained:

[W]hen we look at our own immune responses, we are actually full of -- not full, but we actually have many, many autoreactive B-cells and T-cells in our blood. But in order for autoimmunity to occur, several things need to line up. One is that those autoreactive T-cells need to be preferentially expanded when they are in this competitive state. So the T-cells are competing against other T-cells to be -- to proliferate and expand. And the winners in this expansion would possibly drive an autoimmune response if they are cross-reactive. The problem is that it's not a very easy process to expand and induce autoimmunity.

The other thing is that the autoreactive antigens need to be processed and presented to the immune system if they are T-cells. And although you could pull out peptides that will stimulate self-peptides, peptides that are part of our own body that will stimulate T-cells, these peptides might not be processed and 20 presented to the immune system. So they might never have the ability to stimulate those autoreactive T-cells.

*Id.* at 243.

Additionally, the animal studies relied on by Dr. Steinman were not relevant, in Dr. Maverakis's view, because they involved injection of whole myelin extract, which contains multiple substances, including lipids, that might cause inflammation. Tr. at 246. The studies did not actually specifically demonstrate that phospholipids, such as those purportedly in the Prevnar 13 vaccine, can induce autoimmunity. *Id.* at 244-45. Dr. Maverakis maintained that "there's no evidence experimentally or in humans that lipids themselves are able to induce autoimmunity on their own." *Id.* at 246. He also pointed to the fact that the experimenters used CFA, a very strong adjuvant, which might have confounded the results. *Id.*

Dr. Maverakis also disagreed with Dr. Steinman's attempt to analogize PTS to GBS, stating that they are "entirely separate diseases." Tr. at 247. He explained that in PTS, there is a pathognomonic finding of an hourglass deformity of the nerves, meaning a finding that is not seen in any other disease, including GBS. *Id.* at 248. Furthermore, PTS is, in 10% of cases, a hereditary illness, caused by the Septin-9 gene. *Id.* This gene is not associated with GBS. *Id.* Because PTS and GBS are different diseases, there is no basis to conclude their pathogenesis would be the same. *Id.* at 249. They are, however, both "neurological conditions that have an autoimmune basis or an autoimmune theory as to why they are caused." *Id.* at 265.

Dr. Maverakis disputed the theory that cytokines such as IL-1 and IL-18 could cause PTS. Tr. at 250-51. IL-1 cytokines are involved in diseases that produce symptoms such as high fevers and neutrophils, unlike what occurs in GBS. *Id.* at 252. While there will be some benefit in inhibiting such cytokines "in the setting of autoimmunity," they are not the drivers of autoimmune diseases. *Id.* at 253. Also, the study Dr. Steinman cited demonstrating IL-18 was upregulated in GBS was "very old"; a more recent paper using more modern technology found the opposite. *Id.* at 253-54 (citing Pier Paolo Sainaghi et al., *The expression pattern of inflammatory mediators in cerebrospinal fluid differentiates Guillain–Barré syndrome from chronic inflammatory demyelinating polyneuropathy*, 51 CYTOKINE 138 (2010) (Ex. G, Tab 10) ("Sainaghi")). In rebuttal, Dr. Steinman defended the older study, Jander & Stoll, pointing out that it used several different test methods and provided "sound and reliable" data. *Id.* at 293-96; Jander & Stoll.

As to onset, Dr. Maverakis opined that the article cited by Dr. Steinman supported an onset of symptoms no more than 21 days following vaccination. Tr. at 255 (citing Tsairis). Tsairis reported a possible onset of more than 28 days from the development of pain to the start of muscle weakness, not from exposure to initial onset. *Id.* Dr. Maverakis conceded on cross that his opinion differs from the timeframe for onset of PTS in the Vaccine Injury Table. *Id.* at 267.

With respect to diagnosis, Dr. Maverakis acknowledged on cross that Petitioner was diagnosed with PTS by a neurologist, but he testified that an “anchoring bias” likely confounded the diagnosis, because Petitioner reported an existing “history” of PTS to the neurologist. Tr. at 272-73. Dr. Maverakis did not believe Petitioner met the criteria for PTS as reflected in the literature. *Id.* at 273-74. He did not have any sensory abnormalities, which are commonly seen in PTS. *Id.* at 275. He was in the wrong age group for PTS, and he did not present with neuropathic pain. *Id.* at 275-76. Dr. Maverakis explained:

And when it comes to making a diagnosis of a rare disease, one of the things that we are taught in clinical informatics is that an uncommon presentation of a common disease is much more likely than a common presentation of an uncommon disease. In this case, the Petitioner had an uncommon disease and an uncommon presentation, making it not more likely than not that he had PTS.

*Id.* at 275.

#### IV. APPLICABLE LAW

##### A. Petitioner’s Burden in Vaccine Program Cases

Under the Vaccine Act, a petitioner may prevail in one of two ways. First, he may show that he suffered a Table injury within the time provided in the Table. § 11(c)(1)(C)(i). “In such a case, causation is presumed.” *Capizzano v. Sec’y of Health & Hum. Servs.*, 440 F.3d 1317, 1320 (Fed. Cir. 2006); *see* § 13(a)(1)(B). Second, where the alleged injury is not listed in the Table, he may demonstrate that he suffered an “off-Table” injury that was caused-in-fact by his vaccination. § 11(c)(1)(C)(ii).

For both Table and non-Table claims, Vaccine Program petitioners bear a “preponderance of the evidence” burden of proof. § 13(a)(1). That is, a petitioner must offer evidence that leads the “trier of fact to believe that the existence of a fact is more probable than its nonexistence before [he] may find in favor of the party who has the burden to persuade the judge of the fact’s existence.” *Moberly v. Sec’y of Health & Hum. Servs.*, 592 F.3d 1315, 1322 n.24 (Fed. Cir. 2010); *see also Snowbank Enter. v. United States*, 6 Cl. Ct. 476, 486 (1984) (mere conjecture or speculation is insufficient under a preponderance standard). The petitioner must demonstrate that the vaccine was “not only [the] but-for cause of the injury but also a substantial factor in bringing about the injury.” *Moberly*, 592 F.3d at 1321 (quoting *Shyface v. Sec’y of Health & Hum. Servs.*, 165 F.3d 1344, 1352 (Fed. Cir. 1999)); *Pafford v. Sec’y of Health & Hum. Servs.*, 451 F.3d 1352, 1355 (Fed. Cir. 2006). A petitioner may not receive a Vaccine Program award based solely on his assertions; rather, the petition must be supported by either medical records or the opinion of a competent physician. § 13(a)(1).

In attempting to establish entitlement to a Vaccine Program award of compensation for a non-Table claim, a petitioner must satisfy all three of the elements established by the Federal Circuit in *Althen v. Secretary of Health and Human Services*. 418 F.3d 1274 (Fed. Cir. 2005).

*Althen* requires a petitioner to establish by preponderant evidence that the vaccination caused his injury “by providing: (1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of a proximate temporal relationship between vaccination and injury.” *Id.* at 1278.

Each of the *Althen* prongs requires a different showing. Under *Althen* prong one, a petitioner must provide a “reputable medical theory” demonstrating that the vaccine received *can cause* the type of injury alleged. *Pafford*, 451 F.3d at 1355-56 (citations omitted). To satisfy this prong, a petitioner’s theory must be based on a “sound and reliable medical or scientific explanation.” *Knudsen v. Sec’y of Health & Hum. Servs.*, 35 F.3d 543, 548 (Fed. Cir. 1994) (citations omitted). Such a theory must only be “legally probable, not medically or scientifically certain.” *Id.* at 548-49; *Bunting v. Sec’y of Health & Hum. Servs.*, 931 F.2d 867, 873 (Fed. Cir. 1991).

A petitioner may satisfy the first *Althen* prong without resort to medical literature, epidemiological studies, demonstration of a specific mechanism, or a generally accepted medical theory. *Andreu v. Sec’y of Health & Hum. Servs.*, 569 F.3d 1367, 1378-79 (Fed. Cir. 2009) (citing *Capizzano*, 440 F.3d at 1325-26). Despite their expertise, special masters are not empowered by statute to conclusively resolve what are complex scientific and medical questions, and thus scientific evidence offered to establish *Althen* prong one is viewed “not through the lens of the laboratorian, but instead from the vantage point of the Vaccine Act’s preponderant evidence standard.” *Id.* at 1380. However, this does not negate or reduce a petitioner’s ultimate burden to establish his entitlement to compensation by preponderant evidence. *W.C. v. Sec’y of Health & Hum. Servs.*, 704 F.3d 1352, 1356 (Fed. Cir. 2013) (citations omitted).

The second *Althen* prong requires proof of a logical sequence of cause and effect, usually supported by facts derived from a petitioner’s medical records. *Althen*, 418 F.3d at 1278; *Andreu*, 569 F.3d at 1375-77; *Capizzano*, 440 F.3d at 1326 (stating that “medical records and medical opinion testimony are favored in vaccine cases, as treating physicians are likely to be in the best position to determine whether a ‘logical sequence of cause and effect show[s] that the vaccination was the reason for the injury’”) (quoting *Althen*, 418 F.3d at 1280). Medical records are generally viewed as particularly trustworthy evidence because they are created contemporaneously with the treatment of the patient. *Cucuras v. Sec’y of Health & Hum. Servs.*, 993 F.2d 1525, 1528 (Fed. Cir. 1993). However, the existence of medical records and/or statements of treating physician views does not require the special master to adopt their conclusions *per se*. § 13(b)(1) (providing that “[a]ny such diagnosis, conclusion, judgment, test result, report, or summary shall not be binding on the special master or court”); *Snyder v. Sec’y of Health & Hum. Servs.*, 88 Fed. Cl. 706, 746 n.67 (2009) (“[T]here is nothing . . . that mandates that the testimony of a treating physician is sacrosanct—that it must be accepted in its entirety and cannot be rebutted.”). As with expert testimony offered to establish a theory of causation, the opinions or diagnoses of treating physicians are only as trustworthy as the reasonableness of their suppositions or bases. The views of treating physicians should also be weighed against other, contrary evidence in the record—including conflicting opinions among such individuals. *Hibbard v. Sec’y of Health & Hum. Servs.*, 100 Fed. Cl. 742, 749 (2011) (it was not arbitrary or capricious for special master to weigh competing treating physicians’ conclusions against each other), *aff’d*, 698 F.3d 1355 (Fed. Cir.

2012); *Caves v. Sec'y of Health & Hum. Servs.*, No. 06-522V 2011 WL 1935813, \*17 (Fed. Cl. Spec. Mstr. Apr. 29, 2011), *mot. for review den'd*, 100 Fed. Cl. 344, 356 (2011), *aff'd without opinion*, 475 Fed. App'x 765 (Fed. Cir. 2012).

The third *Althen* prong requires establishing a “proximate temporal relationship” between the vaccination and the injury alleged. *Althen*, 418 F.3d at 1281. That term has been equated to the phrase “medically acceptable temporal relationship.” *Id.* Thus, a petitioner must offer “preponderant proof that the onset of symptoms occurred within a timeframe which, given the medical understanding of the disorder’s etiology, it is medically acceptable to infer causation.” *de Bazan v. Sec'y of Health & Hum. Servs.*, 539 F.3d 1347, 1352 (Fed. Cir. 2008). The explanation for what is a medically acceptable timeframe must also be consistent with the theory for how the relevant vaccine can cause the alleged injury (*Althen* prong one’s requirement). *Id.* at 1352; *Shapiro v. Sec'y of Health & Hum. Servs.*, 101 Fed. Cl. 532, 542 (2011), *recons. denied after remand on other grounds*, 105 Fed. Cl. 353 (2012), *aff'd without op.*, 503 F. App'x 952 (Fed. Cir. 2013); *Koehn v. Sec'y of Health & Hum. Servs.*, No. 11-355V, 2013 WL 3214877 (Fed. Cl. Spec. Mstr. May 30, 2013), *aff'd*, 773 F.3d 1239 (Fed. Cir. 2014).

## B. Law Governing Analysis of Fact Evidence

The process for making factual determinations in Vaccine Program cases begins with analyzing the medical records, which are required to be filed with the petition. §11(c)(2). The special master is required to consider “all [] relevant medical and scientific evidence contained in the record,” including “any diagnosis, conclusion, medical judgment, or autopsy or coroner’s report which is contained in the record regarding the nature, causation, and aggravation of the petitioner’s illness, disability, injury, condition, or death,” as well as the “results of any diagnostic or evaluative test which are contained in the record and the summaries and conclusions.” §13(b)(1)(A). The special master is then required to weigh the evidence presented, including contemporaneous medical records and testimony. *See Burns v. Sec'y of Health & Hum. Servs.*, 3 F. 3d 415, 417 (Fed. Cir. 1993) (it is within the special master’s discretion to determine whether to afford greater weight to contemporaneous medical records than to other evidence, such as oral testimony surrounding the events in question that was given at a later date, provided that such determination is based on a rational analysis).

Medical records created contemporaneously with the events they describe are generally trustworthy, because they “contain information supplied to or by health professionals to facilitate diagnosis and treatment of medical conditions,” where “accuracy has an extra premium.” *Kirby v. Sec'y of Health & Hum. Servs.*, 997 F.3d 1378, 1382 (Fed. Cir. 2021) (quoting *Cucuras*, 993 F.2d at 1528). Accordingly, if the medical records are clear, consistent, and complete, then they should be afforded substantial weight. *See generally Lowrie v. Sec'y of Health & Hum. Servs.*, No. 03-1585V, 2005 WL 6117475 at \*19 (Fed. Cl. Spec. Mstr. Dec. 12, 2005). Indeed, contemporaneous medical records are often found to be deserving of greater evidentiary weight than oral testimony, especially where such testimony conflicts with the record evidence. *Cucuras*, 993 F.2d at 1528; *see also Murphy v. Sec'y of Health & Hum. Servs.*, 23 Cl. Ct. 726, 733 (1991), *aff'd per curiam*, 968 F.2d 1226 (Fed. Cir. 1992), *cert. den'd*, *Murphy v. Sullivan*, 506 U.S. 974 (1992) (citing *United States v. U.S. Gypsum Co.*, 333 U.S. 364, 396 (1947) (“[i]t has generally been held that oral testimony which is in conflict with contemporaneous documents is entitled to

little evidentiary weight.”)).

However, there are situations in which compelling oral testimony could be more persuasive than written medical records, such as where records are deemed to be incomplete or inaccurate. *Campbell v. Sec'y of Health & Hum. Servs.*, 69 Fed. Cl. 775, 779 (2006) (“[L]ike any norm based upon common sense and experience, this rule should not be treated as an absolute and must yield where the factual predicates for its application are weak or lacking.”); *Lowrie*, 2005 WL 6117475, at \*19 (“Written records which are, themselves, inconsistent, should be accorded less deference than those which are internally consistent.”) (quoting *Murphy*, 23 Cl. Ct. at 733)). Ultimately, the special master should assess each witness’s credibility when determining the weight their testimony should be afforded. *Andreu*, 569 F.3d at 1379; *Bradley v. Sec'y of Health & Hum. Servs.*, 991 F.2d 1570, 1575 (Fed. Cir. 1993).

In determining the accuracy and completeness of medical records, the Court of Federal Claims has listed four possible explanations for inconsistencies between contemporaneously created medical records and later testimony: (1) a person’s failure to recount to the medical professional everything that happened during the relevant time period; (2) the medical professional’s failure to document everything reported to her or him; (3) a person’s faulty recollection of the events when presenting testimony; or (4) a person’s purposeful recounting of symptoms that did not exist. *LaLonde v. Sec'y of Health & Hum. Servs.*, 110 Fed. Cl. 184, 203-04 (2013), *aff'd*, 746 F.3d 1334 (Fed. Cir. 2014). In deciding whether to afford greater weight to contemporaneous medical records or other evidence, such as testimony, a rational analysis must be explicated. *Burns*, 3 F.3d at 417.

### C. Analysis of Expert Testimony

Establishing a sound and reliable medical theory connecting the vaccine to the injury often requires a petitioner to present expert testimony in support of his or her claim. *Lampe v. Sec'y of Health & Hum. Servs.*, 219 F.3d 1357, 1361 (Fed. Cir. 2000). Vaccine Program expert testimony is usually evaluated according to the factors for analyzing scientific reliability set forth in *Daubert v. Merrell Dow Pharm., Inc.*, 509 U.S. 579, 594-96 (1993). See *Cedillo v. Sec'y of Health & Hum. Servs.*, 617 F.3d 1328, 1339 (Fed. Cir. 2010) (citing *Terran v. Sec'y of Health & Hum. Servs.*, 195 F.3d 1302, 1316 (Fed. Cir. 1999)). “The *Daubert* factors for analyzing the reliability of testimony are: (1) whether a theory or technique can be (and has been) tested; (2) whether the theory or technique has been subjected to peer review and publication; (3) whether there is a known or potential rate of error and whether there are standards for controlling the error; and (4) whether the theory or technique enjoys general acceptance within a relevant scientific community.” *Terran*, 195 F.3d at 1316 n.2 (citing *Daubert*, 509 U.S. at 592-95).

The *Daubert* factors play a slightly different role in Vaccine Program cases than they do in other federal judicial proceedings. Those factors are employed by judges to exclude evidence that is unreliable and potentially confusing to a jury. In Vaccine Program cases, these factors are generally used to assess the reliability and weight of scientific evidence. *Davis v. Sec'y of Health & Hum. Servs.*, 94 Fed. Cl. 53, 66-67 (2010) (“[U]niquely in this Circuit, the *Daubert* factors have been employed also as an acceptable evidentiary-gauging tool with respect to persuasiveness of expert testimony already admitted[.]”). The flexible use of the *Daubert* factors to evaluate

persuasiveness and reliability of expert testimony has routinely been upheld. *See, e.g., Snyder*, 88 Fed. Cl. at 743.

Respondent frequently offers one or more experts of his own to rebut a petitioner's case. Where both sides offer expert testimony, a special master's decision may be "based on the credibility of the experts and the relative persuasiveness of their competing theories." *Broekelschen v. Sec'y of Health & Hum. Servs.*, 618 F.3d 1339, 1347 (Fed. Cir. 2010) (citing *Lampe*, 219 F.3d at 1362). Nothing requires the acceptance of an expert's conclusion "connected to existing data only by the *ipse dixit* of the expert," especially if "there is simply too great an analytical gap between the data and the opinion proffered." *Snyder*, 88 Fed. Cl. at 743 (quoting *Gen. Elec. Co. v. Joiner*, 522 U.S. 136, 146 (1997)). A "special master is entitled to require some indicia of reliability to support the assertion of the expert witness." *Moberly*, 592 F.3d at 1324. Weighing the relative persuasiveness of competing expert testimony, based on a particular expert's credibility, is part of the overall reliability analysis special masters must employ in Vaccine Program cases. *Id.* at 1325-26 ("[a]ssessments as to the reliability of expert testimony often turn on credibility determinations"); *see also Porter v. Sec'y of Health & Hum. Servs.*, 663 F.3d 1242, 1250 (Fed. Cir. 2011) ("[T]his court has unambiguously explained that special masters are expected to consider the credibility of expert witnesses in evaluating petitions for compensation under the Vaccine Act").

#### **D. Consideration of Medical Literature**

Finally, although this decision discusses some but not all the medical literature in detail, I have reviewed and considered all the medical records and literature submitted in this matter. *See Moriarty v. Sec'y of Health & Hum. Servs.*, 844 F.3d 1322, 1328 (Fed. Cir. 2016) ("We generally presume that a special master considered the relevant record evidence even though [s]he does not explicitly reference such evidence in h[er] decision."); *Simanski v. Sec'y of Health & Hum. Servs.*, 115 Fed. Cl. 407, 436 (2014) ("[A] Special Master is 'not required to discuss every piece of evidence or testimony in her decision.'") (citation omitted), *aff'd*, 601 F. App'x 982 (Fed. Cir. 2015).

### **V. ANALYSIS**

#### **A. Diagnosis**

As a threshold matter, a petitioner must establish that he suffered the injury for which he seeks compensation. *Broekelschen*, 618 F.3d at 1346. "The function of a special master is not to 'diagnose' vaccine-related injuries, but instead to determine 'based on the record as a whole and the totality of the case, whether it has been shown by a preponderance of the evidence that a vaccine caused the [petitioner]'s injury.'" *Andreu*, 569 F.3d at 1382 (quoting *Knudsen*, 35 F.3d at 549). "Although the Vaccine Act does not require absolute precision, it does require the petitioner to establish an injury – the Act specifically creates a claim for compensation for 'vaccine-related injury or death.'" *Stillwell v. Sec'y of Health & Hum. Servs.*, 118 Fed. Cl. 47, 56 (2014) (quoting 42.U.S.C. § 300aa-11(c)). Accordingly, the Federal Circuit has concluded that it is "appropriate for the special master to first determine what injury, if any, [is] supported by the evidence presented

in the record” before applying a causation analysis pursuant to *Althen. Lombardi v. Sec'y of Health & Hum. Servs.*, 656 F.3d 1343, 1351-53 (Fed. Cir. 2011).

### 1. Parsonage Turner Syndrome

PTS is a fairly rare condition, occurring in about 1 in 1,000 people annually. See Nes van Alfen & Baziel G.M. van Engelen, *The clinical spectrum of neuralgic amyotrophy in 246 cases*, 129 BRAIN 438, at 438 (2006) (Ex. H, Tab 19) (“van Alfen (2006)”). In its classic form, it presents with “abrupt onset of shoulder pain, usually unilaterally, followed by progressive neurological deficits of motor weakness, dysesthesias, and numbness.” Feinberg & Radecki at 199. Acute pain is the first symptom in 90% of PTS patients. Clemens Gstoettner et al., *Neuralgic amyotrophy: a paradigm shift in diagnosis and treatment*, 91 J. NEUROL NEUROSURGERY PSYCHIATRY 879, 881 (2020) (Ex. G, Tab 1) (“Gstoettner”). The presenting pain in PTS

may begin insidiously but quickly amplifies in severity and intensity. The acute period of pain is subsequently replaced over a course of a few days to weeks with progressive weakness, reflex changes, and sensory abnormalities in varying presentations that typically involve the shoulder girdle musculature and proximal upper limb muscles.

Feinberg & Radecki at 199. The pain is constant and severe and might interfere with sleep. *Id.* at 200; *see also* Schreiber at 416 (PTS pain “is reported as ‘constant’ in the first several weeks” and is often described as “sharp” or “throbbing”). It might reach levels of 7-10 on the Visual Analogue Scale. Gstoettner at 881. In most cases, the “pain radiates from the cervical spine or shoulder region into the arm.” *Id.* The “duration of pain is almost always self-limiting, lasting 1 to 2 weeks, but on rare occasions persisting for longer periods of time.” Feinberg & Radecki at 200.

The onset of muscle weakness occurs within two weeks “in the majority of patients.” Gstoettner at 881. In classic PTS, this will commonly affect “upper plexus nerves such as the suprascapular, the long thoracic, the musculocutaneous or the axillary nerve, most frequently in some combination.” *Id.* Sensory symptoms such as numbness and paresthesia are also present in most cases. *Id.*

PTS usually has a self-limiting clinical course and rarely becomes a chronic condition. About 75-80% of patients recover within 2 years, and 90% recover within 3 years. First Maverakis Rep. at 8; Tsairis at 117 (reviewing the history of 99 PTS patients). Such a recovery is expected “regardless of the duration, subsequent course, location, and severity of the plexus lesion.” Tsairis at 117.

PTS is associated with several risk factors, including infection, strenuous exercise, including weightlifting, and surgery. Gstoettner at 880; Nata Parnes et al., *Atypical Pectoralis Major Muscle Wasting in a Recreational Weight Lifter*, 39 ORTHOPEDICS e756, e756 (2016) (Ex. D, Tab 9) (“Parnes”); Feinberg & Radecki at 200 (Table 1). The Vaccine Injury Table includes the injury of PTS/brachial neuritis if the condition’s onset is within 2-28 days following vaccination with a tetanus toxoid-containing vaccine. 42 C.F.R. § 100.3(a)(I).

## 2. Rotator Cuff Arthropathy

A rotator cuff tear in combination with arthritis is termed a “rotator cuff tear arthropathy.” Abrams Rep. at 4. Rotator cuff pathologies are common, with full-thickness tears affecting as many as 50% of patients over the age of 60. *Id.* (citing Ken Yamaguchi et al., *The Demographic and Morphological Features of Rotator Cuff Disease*, 88-A J. OF BONE & JOINT SURGERY 1699 (2006) (Ex. H, Tab 1) (“Yamaguchi”)); *see also* Carr Rep. at 6; Tr. at 198 (Dr. Abrams’s testimony). Rotator cuff arthritis is also very common, especially in older patients. Abrams Rep. at 4; Claudio Chillemi & Vincenzo Franceschini, *Shoulder Osteoarthritis*, 2013 ARTHRITIS 1, 1 (2012) (Ex. H, Tab 2) (“Chillemi”) (up to 32.8% of patients over 60 have osteoarthritis of the shoulder) (up to 32.8% of patients over 60 have osteoarthritis of the shoulder).

The majority of rotator cuff tears remain asymptomatic for some time but become symptomatic as tears enlarge. Tr. at 200 (Dr. Abrams’s testimony); *see also* Rebekah L. Lawrence et al., *Asymptomatic Rotator Cuff Tears*, 9 JBJS REV. 1, 6 (2019) (Ex. G, Tab 6) (“Lawrence”) (reporting that 23-51% of patients with asymptomatic rotator cuff tears develop pain within 1.5 to 3 years). Sometimes symptoms appear without an identifiable triggering event. Tr. at 200 (Dr. Abrams’s testimony). The risk of symptoms developing increases with age. *Id.* at 136-37 (Dr. Carr’s testimony). Also, vigorous exercise such as weightlifting is a risk factor for rotator cuff arthritis and for symptomatic rotator cuff tears. Abrams Rep. at 4; First Leist Rep. at 6; First Maverakis Rep. at 9; Carr Rep. at 6-7.

## 3. Petitioner’s condition is not consistent with classic PTS.

The parties dispute whether Petitioner had PTS, as alleged. I conclude that Petitioner has failed to preponderantly establish he developed PTS. As Respondent’s experts persuasively opined, Petitioner’s initial post-vaccination symptoms and subsequent course were inconsistent with classic PTS but were consistent with rotator cuff arthropathy.

The evidence does not show Petitioner initially experienced the onset of the searing, unrelenting neck and/or shoulder pain that occurs in 90% of PTS cases. *See* Abrams Rep. at 8; First Leist Rep. at 6; Gstoettner at 881; Feinberg & Radecki at 199; Tr. at 193-94 (Dr. Abrams testifying that his PTS patients have “exclusively” presented with fairly severe shoulder pain as their primary complaint). There are several different accounts of Petitioner’s first symptoms. In his affidavit, he stated that in December 2014, he experienced “pain and discomfort” in his neck, right shoulder, and right arm. Pet. Affidavit at 1. At the hearing, he testified that he developed pain, “soreness,” and “stiffness” in his neck and shoulder on December 20 or 21, 2014. Tr. at 8-9. Mrs. Parker’s affidavit stated that Petitioner developed “noticeable discomfort” on the right side of his chest, right arm, and right shoulder “around the end of December.” Mrs. Parker Affidavit at 1. At the hearing, though, she testified that within “two to three weeks” of vaccination, or between about December 5 and 12, 2014, Petitioner told her “his shoulder and neck and all down his right arm was kind of like hurting, beginning to hurt.” Tr. at 45. Notably, she and Petitioner both testified that he decided to wait until the end of December to see if the pain resolved before calling his physician; this suggests his pain was not extreme. *Id.* at 9, 45, 68. The record from the initial visit with Dr. Dageforde on April 20, 2015, indicates Petitioner complained of

atrophy of his right biceps and pectoral muscles; he did not report *any* current or resolved pain. Ex. 7 at 8. The record from the first visit with Dr. Hardy on May 28, 2015, indicates he reported having discomfort in his neck only, beginning in January 2015. Ex. 5 at 5. Even a liberal reading of this evidence does not support the conclusion that Petitioner developed the severe, constant pain characteristic of PTS.<sup>11</sup>

Also, while PTS pain typically resolves within one to two weeks and is replaced by weakness, Petitioner reported pain lasting many months, with overlapping weakness/atrophy. *See* Third Leist Rep. at 2 (the “pain associated with [PTS] is almost always self-limiting and normally lasts 1 to 2 weeks,” but Petitioner had chronic pain); Gstoettner at 881. When Petitioner saw Dr. Walker on August 12, 2015, he complained of right-sided shoulder pain that had bothered him for about six months. Ex. 9 at 1. When he saw Dr. Higgs on January 11, 2016, he reported right shoulder pain since July 2015 (again, about six months before). Ex. 4 at 19.

Petitioner’s pattern of muscle weakness and atrophy were also uncharacteristic of PTS. During his initial visit with Dr. Dageforde, he complained of atrophy of “portions of the right biceps and right pectoral muscles.” Ex. 7 at 8. On exam, he exhibited atrophy of the lateral right pectoral muscle and right triceps (not the biceps). *Id.* at 8-9. When he first saw Dr. Hardy on May 28, 2015, he reported wasting of his triceps and pectoral muscles, and weakness and atrophy of those muscles were observed on exam. Ex. 5 at 5-6. As Dr. Abrams persuasively opined, it is quite rare to see atrophy of the pectoral muscles in PTS and even rarer to see atrophy of both the triceps and the pectoral muscles. Abrams Rep. at 4. He explained that two nerves supply the pectoral muscle, including the medial pectoral nerve, and both of those would need to be impaired to create the “gross atrophy” that was observed in Petitioner.<sup>12</sup> Tr. at 217; *see* Ex. L (diagram of the brachial plexus). However, the medial pectoral nerve “is very rarely involved in [PTS].” Tr. at 218. Also, the triceps muscle is innervated by the radial nerve; thus, PTS affecting both the pectoralis and triceps muscles would require “involvement of the lateral pectoral nerve, medial pectoral nerve and radial nerve,” which is not seen in the classic condition.<sup>13</sup> *Id.*; *see also* Nes van

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<sup>11</sup> At the hearing, Petitioner testified on rebuttal that he has a high tolerance for pain and would continue his normal activities even when experiencing pain. Tr. at 287-88. While that might be true, at no point did he describe his pain after vaccination in a manner characteristic of PTS.

<sup>12</sup> In his reply brief, Petitioner argues that Dr. Abrams’s point is incorrect because only the lateral aspect of his right pectoralis muscle was atrophied on exam at his first visit with Dr. Dageforde. Pet.’s Reply Br. at 4; *see* Ex. 7 at 8-9 (documenting that the “lateral right pectoral muscle[] appears atrophied”). He contends that this shows Petitioner’s condition only affected his lateral pectoral nerve, not his medial pectoral nerve, making a PTS diagnosis appropriate. Pet.’s Reply Br. at 4. He does not cite any expert or medical opinion in support of this conclusion, however. Without such evidence, I cannot conclude that atrophy of the “lateral right pectoral muscle” necessarily means only the lateral pectoral nerve was affected, nor that PTS would be a proper diagnosis if that were the case.

<sup>13</sup> Dr. Steinman cited the Schreiber case report, in which a patient was diagnosed with PTS after presenting with neck pain. Second Steinman Rep. at 1. I note that the EMG of the patient in Schreiber showed denervation of the serratus anterior and deltoid muscles; the triceps and pectoral muscles were not reported to be affected. Schreiber at 417. Also, unlike Petitioner, that patient did not have a history of musculoskeletal disease. *Id.* Thus, this case report is of limited use to show that Petitioner’s presentation fit the reported characteristics of PTS.

Alfen et al., *Incidence of Neuralgic Amyotrophy (Parsonage Turner Syndrome) in a Primary Care Setting - A Prospective Cohort Study*, 10 PLOS ONE 1, 4 (Table 2) (2015) (Ex. D, Tab 16) (“van Alfen (2015)”) (inclusion criteria for study of PTS included paresis of the long thoracic nerve, suprascapular nerve, or anterior interosseous nerve); Feinberg & Radecki at 201-02 (the upper trunk of the brachial plexus, suprascapular nerve, long thoracic nerve, and axillary nerves are the most commonly involved in PTS, while the ulnar, radial, and median nerves are least commonly involved).

Several other aspects of Petitioner’s condition were not suggestive of PTS. Although 90% of PTS patients recover full functionality within three years, Petitioner was still complaining of right shoulder pain in April 2018, nearly three and a half years after the vaccination. Ex. 66 at 21; see Tsairis at 7 (reporting that 89% of the patients studied had fully recovered within three years). Also, Petitioner was 67 years old when he developed his “PTS” symptoms, but most PTS patients are under the age of 60. Second Maverakis Rep. at 1; see Gstoettner at 885 (reviewing the literature and reporting that the average PTS patient in the studies reviewed was between 21 and 46). His MRI did not show the focal intrinsic nerve constrictions that Dr. Maverakis testified are seen in the “vast majority of patients.” Second Maverakis Rep. at 1; Tr. at 247; Gstoettner at 882 (studies have found that in PTS, “structural nerve alterations are the norm rather than the exception”); Darryl B. Sneag et al., *MRI Bullseye Sign: An Indicator of Peripheral Nerve Construction in Parsonage-Turner Syndrome*, 56 MUSCLE & NERVE 99, 99 (2017) (Ex. G, Tab 3) (“Sneag (2017)”) (MRIs of six PTS patients with “absent or minimal recovery” all showed hourglass nerve constrictions in various nerves).<sup>14</sup>

#### 4. Petitioner’s condition is consistent with symptomatic rotator cuff arthropathy.

The record shows that Petitioner’s symptoms were more likely explained by a pre-existing rotator cuff arthropathy than by PTS. Both parties’ experts agreed that Petitioner had a rotator cuff tear that likely predicated his vaccination. See, e.g., Tr. at 123 (Dr. Carr’s testimony); id. at 210 (Dr. Abrams’s testimony). The experts also agreed that he had rotator cuff arthritis. Id. at 136 (Dr. Carr); id. at 197 (Dr. Abrams’s testimony). These conditions are very common in people Petitioner’s age and in regular weightlifters. In August 2015, Dr. Walker suspected a rotator cuff tear was causing Petitioner’s symptoms, which had been ongoing for six months. Ex. 9 at 1. In September, a full-thickness tear and arthritis were observed on MRI, prompting Dr. Walker to recommend surgical repair of the tear. Id. at 8. Petitioner declined to pursue this option. Tr. at 24-25, 55-56. In January 2016, Dr. Higgs documented chronic shoulder pain and observed weakness with external rotation, which Dr. Abrams opined was “consistent with rotator cuff pathology[,] as the rotator cuff muscles are the only external rotators of the shoulder.” Abrams Rep. at 6; Ex. 4 at 19-20. Petitioner also exhibited scapular dyskinesis, which signifies

<sup>14</sup> Dr. Abrams acknowledged that PTS might present differently than its classic version. See Tr. at 216. But as Dr. Maverakis persuasively pointed out, the principles of clinical informatics suggest it would be very unlikely that a relatively rare disease such as PTS would present atypically in several different respects. Id. at 275 (noting that an uncommon presentation of an uncommon disease is highly unusual). This is especially so in the context of Petitioner’s pre-existing rotator cuff tear and weightlifting practice, which readily fit his symptomology and clinical course.

periscapular dysfunction and is associated with rotator cuff symptoms. Tr. at 201-03. He had a positive impingement sign, also consistent with a symptomatic rotator cuff tear. Abrams Rep. at 7; Ex. 9 at 1 (examination showing grossly positive Neer and Hawkins tests and external rotator weakness); *see Atsushi Yamamoto et al., Factors involved in the presence of symptoms associated with rotator cuff tears: a comparison of asymptomatic and symptomatic rotator cuff tears in the general population*, 20 J. OF SHOULDER & ELBOW SURGERY 1133, 1133 (2011) (Ex. H, Tab 17) (“Yamamoto”) (study showing symptomatic rotator cuff tears present with positive impingement signs, external rotation weakness, and a tear in the dominant arm). In sum, Petitioner’s long-lasting shoulder pain, atrophy, and muscular dysfunction were wholly consistent with rotator cuff arthropathy that became symptomatic after the vaccination. *See, e.g.*, Second Leist Rep. at 1; Sambandam at 1 (noting that, over time, partial rotator cuff tears enlarge and become full-thickness tears, causing the development of “distinct chronic pathological changes due to muscle retraction, fatty infiltration and muscle atrophy”); Tr. at 196 (Dr. Abrams opining that Petitioner had a “classic rotator cuff arthropathy,” which became symptomatic after the vaccination).

5. Petitioner has not proven his “PTS” caused him to develop symptomatic rotator cuff arthropathy.

Petitioner does not dispute that he had rotator cuff arthropathy, but he contends that his condition became symptomatic *because* of his PTS. Dr. Carr opined that after the vaccination, Petitioner developed PTS, which led to atrophy, causing his shoulder musculature to become unbalanced. Carr Rep. at 7-8. This, in turn, caused his rotator cuff to become symptomatic. *Id.* Dr. Carr likened Petitioner’s case to those in which overhead throwing athletes or swimmers develop shoulder pain “when their strength and flexibility become unbalanced” between the posterior and anterior muscles. *Id.* at 7. He explained this imbalance causes the shoulder joint to create an abnormal shearing force during use, eventually leading to inflammation, swelling, pain, and rotator cuff and joint pathology. *Id.*

I conclude that Petitioner failed to show his rotator cuff symptoms were brought about by PTS. Most significantly, as already explained, the evidence does not preponderantly support the foundational claim that Petitioner developed PTS. Furthermore, I do not find persuasive Dr. Carr’s opinion that Petitioner’s “PTS”-related atrophy could lead to rotator cuff symptomology. Dr. Abrams pointed out that there was no literature supporting the hypothesis that weakness of the pectoral or triceps muscles could contribute to imbalance of the shoulder, as Dr. Carr proposed. Tr. at 201. Dr. Carr conceded that he did a literature search and found that “no one has ever reported this before.” *Id.* at 134. Dr. Abrams compared Petitioner’s case to patients he has seen with chronic pectoralis rupture, who develop atrophy of the pectoralis muscle and weakness of internal rotation of the shoulder but do *not* develop shoulder pain. Abrams Rep. at 6; Tr. at 209. This pattern suggests that “the pectoralis kind of muscle tendon unit does not play really any role in this kind of balancing effect of the shoulder.” Tr. at 209-10. He further explained that the pectoralis muscle is responsible for adduction and internal rotation of the humerus, while the rotator cuff muscles are responsible for external rotation. Abrams Rep. at 6. Because Petitioner was observed to have weakness of external rotation on exam, and because the triceps is not known to be involved in symptomatic rotator cuff pathology, this implicates his rotator cuff as the cause of his symptoms, rather than the atrophy of the pectoral and triceps muscles. *Id.*

Dr. Abrams persuasively distinguished Petitioner's presentation from shoulder conditions affecting overhead throwing athletes and swimmers, noting that these conditions involve different patient populations, mechanics, and muscles. Abrams Rep. at 4-6; *see Stephen S. Burkhart et al., The Disabled Throwing Shoulder: Spectrum of Pathology Part I: Pathoanatomy and Biomechanics*, 19 J. OF ARTHROSCOPIC & RELATED SURGERY 404 (2003) (Ex. H, Tab 3) ("Burkhart Part I"); Stephen S. Burkhart et al., *The Disabled Throwing Shoulder: Spectrum of Pathology Part II: Evaluation and Treatment of SLAP Lesions in Throwers*, 19 J. OF ARTHROSCOPIC & RELATED SURGERY 531 (2003) (Ex. H, Tab 4) ("Burkhart Part II"); Stephen S. Burkhart et al., *The Disabled Throwing Shoulder: Spectrum of Pathology Part III: The SICK Scapula, Scapular Dyskinesis, the Kinetic Chain, and Rehabilitation*, 19 J. OF ARTHROSCOPIC & RELATED SURGERY 641 (2003) (Ex. H, Tab 5) ("Burkhart Part III") (three articles showing that GIRD in overhead athletes results from a tight posteroinferior capsule and periscapular muscle weakness, which leads to scapular dyskinesis); Elizabeth Matzkin et al., *Swimmer's Shoulder: Painful Shoulder in Competitive Swimmer*, 24 J. AM. ACAD. ORTHOPEDIC SURGERY 527 (2016) (Ex. H, Tab 6) ("Matzkin") (noting that swimmer's shoulder can be caused by periscapular muscle weakness). Dr. Carr acknowledged these distinctions as well, though he maintained that Petitioner had scapular dyskinesis, "which is exactly the same thing that's talked about in swimmer's and thrower's shoulders." Tr. at 135. Notably, however, Dr. Carr did not opine or cite any literature showing that atrophy of the pectoral/triceps muscles *causes* scapular dyskinesis. Thus, Dr. Carr failed to connect Petitioner's alleged PTS to his rotator cuff symptoms. By contrast, rotator cuff pathology is associated with periscapular muscular issues and scapular dyskinesis. *See* Tr. at 202 (Dr. Abrams's testimony).

Lastly, I note that Petitioner's neurologist, Dr. Hardy, diagnosed him with PTS. Ex. 5 at 5. I am not bound to accept Dr. Hardy's diagnosis, and, as discussed, the overall record supports rotator cuff pathology as the more likely explanation of Petitioner's symptoms. *See* §13(b)(1) (providing that "[a]ny such diagnosis, conclusion, judgment, test result, report, or summary shall not be binding on the special master or court."); *Snyder*, 88 Fed. Cl. at 746 n.67 ("[T]here is nothing . . . that mandates that the testimony of a treating physician is sacrosanct—that it must be accepted in its entirety and cannot be rebutted."); *Eloyan v. Sec'y of Health & Hum. Servs.*, No. 18-1450V, 2023 WL 9053983, at \*8 (Fed. Cl. Nov. 17, 2023), *motion for relief from judgment denied*, No. 18-1450V, 2025 WL 1291260 (Fed. Cl. Mar. 25, 2025). Moreover, Dr. Hardy did not order any testing, such as an EMG, to confirm his PTS diagnosis or rule out other conditions, despite Petitioner's risk factors and presentation supporting a possible rotator cuff arthropathy. Dr. Steinman agreed further evaluation was warranted in Petitioner's case. Third Steinman Rep. at 1; *see also* Darryl B. Sneag et al., *Brachial Plexitis or Neuritis? MRI Features of Lesion Distribution in Parsonage-Turner Syndrome*, 58 MUSCLE & NERVE 359, 359 (2018) (Ex. G, Tab 2) ("Sneag (2018)") (noting that MRI and electrodiagnostic testing are important for diagnosing and characterizing PTS). Given that the later MRI did show rotator cuff arthropathy but did not show any focal nerve constrictions, this omission by Dr. Hardy was significant. His PTS diagnosis also was not confirmed by another neurologist, unlike the rotator cuff pathology, which was diagnosed by two orthopedists.<sup>15</sup>

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<sup>15</sup> Although both Drs. Walker and Higgs discussed PTS in Petitioner's history, neither orthopedist independently confirmed that diagnosis. Ex. 4 at 19-20; Ex. 9 at 1.

## B. Causation

Although I conclude Petitioner has failed to prove the diagnosis alleged, I nonetheless will address the evidence relating to causation. I determine that Petitioner has not met his burden of proof that the subject vaccine could or did cause him to develop PTS.

### 1. Althen Prong One

In the context of the Program, “to establish causation, the standard of proof is preponderance of evidence, not scientific certainty.” *Langland v. Sec'y of Health & Hum. Serv.*, 109 Fed. Cl. 421, 441 (Fed Cir. 2013). Petitioner’s burden under *Althen*’s first prong is to provide a medical theory causally connecting the vaccination and the injury. *Id.* This theory must be sound and reliable. *Boatman v. Sec'y of Health & Hum. Servs.*, 941 F.3d 1351, 1359 (Fed. Cir. 2019).

Petitioner did not present any epidemiologic or case report evidence of an association between PTS and Prevnar 13 vaccination. Dr. Steinman admitted he did not have any such evidence, conceding that the pathogenesis of PTS has not been well-studied. Tr. at 95-96; First Steinman Rep. at 7. Nor did Petitioner produce any literature directly proposing a hypothetical mechanism by which the Prevnar 13 vaccine could cause PTS.

#### a. Molecular Mimicry

Dr. Steinman advanced two separate theories of causation. The first was predicated on molecular mimicry between myelin phospholipids purportedly involved in PTS-like diseases and components of the vaccine. Tr. at 79-80. He extrapolated from two animal studies, Brostoff and Wisniewski, which he argued supported the notion that PTS is caused by an autoimmune response to phospholipids in peripheral nerve myelin. First Steinman Rep. at 7. In Brostoff, published in 1973, guinea pigs were injected with whole brachial plexus nerve myelin combined with CFA. Brostoff at 502. In some animals, this induced non-symptomatic inflammatory lesions in the brachial plexus. *Id.* In the 1974 Wisniewski study, the investigators induced recurrent experimental allergic polyganglioradiculoneuritis in monkeys injected with peripheral nerve myelin and CFA. Wisniewski at 347. As Dr. Maverakis pointed out, however, because these older studies used whole myelin, which contains multiple substances that could be inflammatory, they failed to support Dr. Steinman’s specific hypothesis that phospholipids within the myelin can induce PTS. Tr. at 244-46. Also, the use of CFA, which is highly immunogenic, confounded the results. *Id.* at 246.

Additionally, Dr. Steinman conceded he had no evidence that antiphospholipid antibodies have ever been found in PTS patients. Tr. at 100-01. He instead relied on the 1993 Gilburd study, in which antibodies to phospholipids were found in GBS patients. First Steinman Rep. at 7; Gilburd at 23. But as Dr. Maverakis explained, antibodies to lipids are common in the general population and are not specific for autoimmunity. Tr. at 241. In fact, the Gilburd authors themselves surmised that the antibodies they detected were “probably produced as a result of the myelin damage rather than the cause of the demyelination.” Gilburd at 23; see Tr. at 101-02. Furthermore, Dr. Steinman’s attempt to analogize PTS to GBS was unconvincing. As Drs. Maverakis and Leist explained, GBS and PTS are distinct conditions with different clinical

presentations. Tr. at 162-63, 247-49. PTS is believed to be an axonal process involving significant, or even complete, denervation of the involved muscles, while GBS is typically a demyelinating condition. *See* First Leist Rep. at 5. The diseases also have “strikingly different clinical presentations.” Second Maverakis Rep. at 7. GBS starts with ascending weakness that begins in the legs and spreads to the upper body, while PTS almost always begins with acute pain and usually remains isolated to one upper extremity. *Id.* at 6. Also, GBS presents with pain in only a minority of cases, whereas severe pain is reported in nearly all PTS cases. *Id.*; *see* Shaoli Yao et al., *Pain during the acute phase of Guillain–Barré syndrome*, 97 MED. 1 (2017) (Ex. G, Tab 9) (“Yao”) (review of GBS cases reporting that only 34.5% of patients report pain during the acute phase of GBS). And, as noted above, PTS has distinctive findings on MRI, which are not seen in GBS.<sup>16</sup> Second Maverakis Rep. at 1.

*b. Aluminum adjuvant-induced cytokines*

Dr. Steinman’s second proposed theory of causation was that the aluminum adjuvant in the Prevnar 13 vaccine stimulates the production of inflammatory cytokines that drive PTS. Again, he had no data suggesting that the cytokines he identified, IL-1 and IL-18, are elevated in PTS patients. He instead relied on data showing the elevation of IL-1 and IL-18 in GBS, a materially different disease, as well as animal studies in EAN models. Jander & Stoll at 253 (2000 study finding significantly elevated serum levels of IL-18 in GBS patients and increased production during active disease progression in EAN models); Nyati at 3540 (elevated IL-1 was observed in GBS patients during disease progression compared to controls); Yu at 614 (anti-IL-18 antibody therapy ameliorates EAN in animals). Dr. Maverakis persuasively pointed out that IL-1 is associated with high fevers, high neutrophils, and other symptoms not associated with PTS. Tr. at 252. He also provided more recent research that failed to show elevated IL-18 in GBS patients. Sainaghi at 138 (2010 study of cerebrospinal fluid of GBS patients finding no significant difference in IL-18 levels between patients and controls). Overall, even putting aside the significant differences between PTS and GBS, this evidence was inadequate to reliably substantiate Dr. Steinman’s theory, nor was there any evidence of elevated levels of IL-1 or IL-18 in Petitioner, as Dr. Steinman acknowledged. Tr. at 107.

In sum, Petitioner has failed to provide a sound and reliable theory of causation, and therefore I conclude he has not met *Althen* prong one.

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<sup>16</sup> Dr. Steinman also relied on his own research showing the presence of antiphospholipid antibodies in central nervous system conditions. In Kanter, lipid-specific antibodies were observed in multiple sclerosis (“MS”) patients, and mice with experimental autoimmune encephalitis (“EAE”) developed more severe disease after immunization with lipids. Kanter at 138. Dr. Maverakis testified that the study also found these antibodies in the controls, suggesting they were not drivers of autoimmunity. Tr. at 242. In Ho, anti-lipid antibodies were again found in MS patients, but in animals they were not observed to induce autoimmunity, only to affect disease severity. *Id.* at 106; Ho at 1-2. These studies are inadequate to substantiate the claim that PTS, a peripheral nervous system disease, is mediated by anti-lipid antibodies.

## 2. Althen Prong Two

Petitioner similarly failed to satisfy *Althen* prong two, which requires proof of “a logical sequence of cause and effect showing that the vaccination was the reason for the injury.” *Althen*, 418 F.3d at 1278. The sequence of cause and effect must be “logical” and legally probable, not medically or scientifically certain.” *Andreu*, 569 F.3d at 1380 (quoting *Knudsen*, 35 F.3d at 548-49). A petitioner is not required to show “epidemiologic studies, rechallenge, the presence of pathological markers or genetic disposition, or general acceptance in the scientific or medical communities to establish a logical sequence of cause and effect.” *Capizzano*, 440 F.3d at 1325. Instead, circumstantial evidence and reliable medical opinions may be sufficient to satisfy the second *Althen* prong. *Id.* Further, special masters are expected to consider the views of treating doctors. *Id.* at 1326. Such views are often persuasive because the doctors have direct experience with the patient whom they are diagnosing -- but they are not necessarily dispositive of the causation question. *See McCulloch v. Sec'y of Health & Hum. Servs.*, No. 09-293V, 2015 WL 3640610, at \*20 (Fed. Cl. Spec. Mstr. May 22, 2015).

None of Petitioner’s treating physicians persuasively attributed his condition to the Prevnar 13 vaccination.<sup>17</sup> Exs. 7 at 8; 5 at 5-6; 9 at 1. Moreover, strenuous exercise, including ardent weightlifting, is a known trigger of PTS. Gstoettner at 880; *see* Parnes at e756. Petitioner testified that he had been lifting weights since he was 13 years old. Tr. at 12. After the vaccination, he continued weightlifting four days per week, up to four hours per session. Pet. Second Affidavit at 1; Tr. at 15. By January 2015, he first noticed weakness in his right arm while weight training. *Id.*; Pet. Affidavit at 1. During his initial visit with Dr. Hardy, he reported he began noticing atrophy and weakness in his right triceps and pectoral muscle “when he was doing his ordinary weightlifting.” Ex. 5 at 5. Dr. Hardy described him as an “avid weightlifter” with very hypertrophied muscles throughout his upper body. *Id.* Thus, even assuming Petitioner developed PTS, it was more likely caused by his weightlifting practice than by the subject vaccination. *See, e.g., Doe 11 v. Sec'y of Health & Hum. Servs.*, 601 F.3d 1349, 1357-58 (Fed. Cir. 2010); *Walther v. Sec'y of Health & Hum. Servs.*, 485 F.3d 1146, 1151 (Fed. Cir. 2007) (although a petitioner does not bear the burden of eliminating all alternative causes for her injury, it is appropriate for a special master to consider evidence relating to such alternative causes in assessing the *Althen* prongs). Finally, as discussed below, the onset of his symptoms did not fall within a medically acceptable timeframe after vaccination, further showing the lack of a logical sequence of cause and effect between the vaccination and the alleged injury.

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<sup>17</sup> At Petitioner’s initial visit with Dr. Hardy on May 28, 2015, Dr. Hardy commented that Petitioner’s PTS began “a few months” after a vaccination; he did not state the vaccine caused the condition. Ex. 5 at 5. Nearly four years later, when Petitioner saw Dr. Hardy for syncope he experienced while exercising, Dr. Hardy summarily stated that he saw Petitioner “in 2016” for PTS “caused by a pneumonia vaccine.” Ex. 67 at 1. This inaccurate recitation of Petitioner’s history is not persuasive evidence of causal attribution, especially because this notation was made after Dr. Hardy learned this litigation was ongoing. *See id.*; Ex. 5 at 8 (Dr. Hardy commenting in 2016 that Petitioner had returned to see him to “facilitate a settlement”).

### 3. Althen Prong Three

*Althen* prong three contains two parts. First, a petitioner must establish the “timeframe for which it is medically acceptable to infer causation,” and second, he must demonstrate that the onset of the disease occurred in this period. *Shapiro*, 101 Fed. Cl. at 542-43.

#### a. *Onset of Petitioner’s symptoms*

There is conflicting evidence concerning when Petitioner’s first symptoms began and what those symptoms were. According to the contemporaneous medical records, his symptoms began no earlier than January 2015. At his visit with Dr. Hardy on May 28, 2015, he reported developing neck discomfort in January 2015. Ex. 5 at 5. At his first visit with Dr. Walker on August 12, 2015, he placed the onset of shoulder pain about six months earlier, in February 2015. Ex. 9 at 1. When he first saw Dr. Higgs on January 11, 2016, he reported shoulder pain since about July 2015. Ex. 4 at 19.

Based on the medical records, the experts for both parties opined that the onset of Petitioner’s symptoms was sometime in January 2015. Dr. Steinman opined that Petitioner developed PTS at least 41 days after vaccination (meaning January 1, 2015, at the earliest). First Steinman Rep. at 5; *see also* First Leist Rep. at 5.

By contrast, Petitioner and his wife testified his symptoms began at various times in December 2014. Petitioner testified that he first experienced right arm, neck, and shoulder pain, soreness, and stiffness on December 20 or 21, 2014, 29-30 days after the November 21 vaccination. Tr. at 8-9. His affidavit said he developed “pain and discomfort” in his neck, right shoulder, and right arm in “December 2014.” Pet. Affidavit at 1. Mrs. Parker testified that Petitioner developed pain in his right arm, chest, and shoulder within “two to three weeks” of vaccination, or between December 5 and 12, 2014. Tr. at 45. But her affidavit said he became “noticeably uncomfortable” on the right side of his chest, right arm, and right shoulder “around the end of December 2014.” Mrs. Parker Affidavit at 1.

To overcome the presumption that contemporaneous written medical records are accurate, contrary testimony must be “consistent, clear, cogent, and compelling.” *Blutstein v. Sec’y of Health & Hum. Servs.*, No. 90-2808 V, 1998 WL 408611, at \*5 (Fed. Cl. Spec. Mstr. June 30, 1998). Because of this presumption, “special masters in this Program have traditionally declined to credit later testimony over contemporaneous records.” *Sturdivant v. Sec’y of Health & Hum. Servs.*, No. 07-788V, 2016 WL 552529, at \*15 (Fed. Cl. Spec. Mstr. Jan. 21, 2016); *see, e.g., Stevens v. Sec’y of Health & Hum. Servs.*, No. 90-221V, 1990 WL 608693, at \*3 (Fed. Cl. Spec. Mstr. Dec. 21, 1990); *see also Vergara v. Sec’y of Health & Hum. Servs.*, No. 08-882V, 2014 WL 2795491, at \*4 (Fed. Cl. Spec. Mstr. Jul. 17, 2014) (“Special Masters frequently accord more weight to contemporaneously-recorded medical symptoms than those recorded in later medical histories, affidavits, or trial testimony.”); *see also Cucuras*, 993 F.2d at 1528 (noting that “the Supreme Court counsels that oral testimony in conflict with contemporaneous documentary evidence deserves little weight”); *Kirby*, 997 F.3d at 1382.

Here, the stories relayed in Petitioner's and Mrs. Parker's affidavits and hearing testimony were inconsistent both as to the timing and the nature of Petitioner's post-vaccination symptoms; this evidence was not "consistent, clear, cogent, and compelling." Thus, these accounts are insufficient to override the contemporaneous medical records, none of which supported the notion that Petitioner developed any symptoms in December 2014. I therefore conclude, based on the medical records, that the onset of Petitioner's symptoms was at least 41 days after vaccination.

*b. An onset of PTS 41 days or more after vaccination is not medically appropriate.*

Dr. Steinman opined that the onset of PTS at least 41 days after Prevnar 13 vaccination is medically acceptable. First Steinman Rep. at 15-16. He referenced literature setting forth the appropriate timeframe for the development of GBS following influenza ("flu") vaccination, arguing that GBS was an appropriate surrogate for PTS because "they are both inflammatory diseases of peripheral nerves." *Id.* at 13, 15-16 (citing Schonberger); Tr. at 88-90, 109. He cited an unnamed abstract (which does not appear to have been filed) purportedly outlining certain clinical similarities between GBS and PTS. *Id.* at 13-14. Finally, he relied on the Michotte case report, which he said reported the onset of PTS seven months after a febrile illness. Second Steinman Rep. at 2.

For several reasons, I do not find Dr. Steinman's opinion persuasive. First, as discussed above, PTS and GBS are distinct conditions, making it inapt to use GBS as a comparator for PTS for this purpose. Second, the Michotte case report is not reliable evidence that PTS can have an onset of 41 days or more after vaccination. As Dr. Leist pointed out, Michotte described two cases of PTS, neither of which had an onset of seven months after a febrile illness. Second Leist Rep. at 3. The first case did not involve a febrile illness at all. Michotte at 71-72. In the second case, the patient reported severe pain in the left shoulder, which lasted for a few weeks and then dissipated. *Id.* at 72. The pain was followed by transient weakness but no atrophy. *Id.* Fifteen years after that episode, after a flu-like illness, the patient developed a similar syndrome in the opposite shoulder. *Id.* She then went about seven months without any pain. *Id.* After that period, she developed *another* febrile illness, "after" which "she again experienced aching pain in the right shoulder." *Id.* The time between the illness and the third pain episode was not stated. *Id.* She was eventually diagnosed with a relapsing, sporadic form of PTS. *Id.* Thus, based on the description of this case in the report, the patient did not have a seven-month latency between her febrile illness and the onset of her pain; she had such a gap *between* her second and third PTS episodes.

Third, the literature submitted by the parties does not support Dr. Steinman's position. In Tsairis, which provided a natural history of PTS in 99 patients, the investigators discussed a subset of 14 patients who reported receiving vaccinations prior to the onset of their PTS symptoms. Tsairis at 111. None of those patients reported onset beyond 21 days after vaccination.<sup>18</sup> *Id.*; see Tr. at 111.

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<sup>18</sup> Similarly, in van Alfen (2006), a study of the natural history of 246 PTS cases, the time to onset after an antecedent event was within 7 days in the large majority of cases; only a small minority reported onset more than two weeks after an event. van Alfen (2006) at 443 (Table 7).

Fourth, other special masters have rejected the proposition that a 41-day onset of PTS following vaccination is medically appropriate. *See Greene v. Sec'y of Health & Hum. Servs.*, No. 11-631V, 2019 WL 4072110, at \*19 (Fed. Cl. Spec. Mstr. Aug. 2, 2019) (denying entitlement where PTS began 41 days after receipt of the tetanus-diphtheria vaccine); *Garner v. Sec'y of Health & Hum. Servs.*, No. 15-063V, 2017 WL 1713184, at \*15 (Fed. Cl. Spec. Mstr. Mar. 24, 2017) (denying entitlement where PTS began 45 days after Hepatitis A and B vaccinations). Also, Dr. Steinman's proposed timeframe is well outside the 2-28-day timeframe set forth in the Vaccine Injury Table for the onset of PTS/brachial neuritis following administration of tetanus-containing vaccines. 42 C.F.R. § 100.3(a)(I).

Thus, I conclude that Petitioner has failed to satisfy *Althen* prong three.

## VII. CONCLUSION

Upon careful evaluation of all the evidence submitted in this matter, including the medical records, the testimony, the experts' opinions, and the medical literature, I conclude that Petitioner has not shown by preponderant evidence that he is entitled to compensation under the Vaccine Act. **His petition is therefore DISMISSED. The clerk shall enter judgment accordingly.<sup>19</sup>**

**IT IS SO ORDERED.**

s/ Jennifer A. Shah  
Jennifer A. Shah  
Special Master

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<sup>19</sup> Pursuant to Vaccine Rule 11(a), the parties may expedite entry of judgment by each filing (either jointly or separately) a notice renouncing their right to seek review.